Suid-Afrikaanse Tydskrif vir Geneeskunde
South African Medical Journal

Posbus 643, Kaapstad P.O. Box 643, Cape Town

Kaapstad, 2 November 1957 Weekliks 2s. 6d. | Vol. 31 No. 44 | Cape Town, 2 November 1957 Weekly 2s. 6d.

AGING FROM CEREBRAL VASCULAR DISEASE SEEN FROM THE PSYCHIATRIC POINT OF VIEW*

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It would be too big a task to discuss all the possible psychiatric complications of cerebral vascular disease. Fortunately our subject is coupled with the problem of aging and I can therefore confine myself to the psychiatry of the second half of life.

I propose to deal with this matter under the headings: (1) Involutional psychoses, (2) Presenile psychoses, (3) Arteriosclerotic psychoses and (4) Senile psychoses with special reference to cerebral vascular disease.

INVOLUTIONAL PSYCHOSES

Involutional psychoses are ill-defined, for under this heading various mental diseases of different aetiology, endogenous, endocrinal and arteriosclerotic are grouped together. The meaning of the word involution is not clear either. I suspect that it is one of those Latin words that have changed their meaning after they have slipped somehow into the medical dictionary. The Germans have a better word for it; they speak of *Rückbildungsalter*. Decline would perhaps be the best translation. However, *verba valent usu*. When does this involution, this decline start? It is an arbitrary matter and depends on which function is referred to. As regards heart and muscles, one is on the decline at 30 when one is a rugby player; as regards learning by heart and photographic memory one may have started one's decline earlier; as regards adaptability to new circumstances, in other words as regards mental elasticity, involution may be considered to start in the early forties.

One can certainly not equate involution with the menopause as is so often done. That would in the first place exclude the male sex, but it would also ignore the fact that women need not be on the decline apart from their productiveness, and are in fact often in the prime of life, after their menses have ceased. It is of course true that many women have nervous complaints during the menopausal period and also that psychoses which occur at that time often show exogenous symptoms like schizoid traits, peculiar paranoid pictures and hallucinations. This simply shows that these psychoses have not only a temperamental but also an exogenous, *in casu* a hormonic aetiology.

It is obvious that the involutional age is different for different organs and for different functions; it certainly varies in different persons. As we are dealing with psychiatry, I want to stress the middle-age feature of decreased mental elasticity and increased secondary function (i.e. the influences of past experiences on our thinking and feeling). It explains why, for instance, true paranoia, previously latent, becomes manifest at the age of 40, why attacks of depression become more persistent and of longer duration at middle-age. It is the chief cause of so-called involutional melancholia.

Those of us who have learned their psychiatry in the later stages of the Kraepelinian era know the endless controversy concerning involutional melancholia being a separate entity or belonging to the manic-depressive psychoses. Whereas depressive phases of the manic-depressive psychosis were curable and followed by a completely normal interval, the involutional melancholia (usually precipitated by a personal loss) presented a somewhat different picture and was more fixed and protracted and could end in permanent mental sickness and dementia. One usually assumed that in the latter case cerebral arteriosclerosis had supervened.

This problem has assumed a new aspect since the advent of electro-convulsive therapy, for it is exactly in involutional melancholia that E.C.T. has recorded its greatest successes. The results of electroconvulsive therapy have in my opinion proved that involutional melancholia is essentially endogenous, not based on organic changes (hormonic or arteriosclerotic) but psychologically explainable, that it is in fact the middle-age reaction in certain predisposed people to personal, seemingly catastrophic changes in life. The loss of mental elasticity prevents the middle-aged person from adapting himself to the altered circumstances and starting a new life.

It must, however, be admitted that some cases of involutional melancholia are complicated by cerebral arteriosclerosis. The diagnosis of early cerebral arteriosclerosis is by no

* A paper read in plenary session at the South African Medical Congress, Durban, September 1957.

means an easy matter. It is a well-known fact that the abscence of arteriosclerosis in other parts of the body does not exclude cerebral arteriosclerosis and *vice versa*. The so-called neurasthenic stage of early cerebral arteriosclerosis, the lack of concentration, the increased mental fatiguability, the slight loss of memory, may well be reversible melancholic signs. The interpretation of retinal vascular changes is not easy either, and in my experience often misleading.

I should like to make a plea for careful E.C.T. in these cases of involutional melancholia with suspected early cerebral arteriosclerosis. It has been my experience that with the newer *Glissando* method and with generous spacing of one's treatments confusion and gross memory defects can be avoided and excellent results achieved. Obviously I would never advocate any form of electrical treatment in cases of pronounced cerebral arterioclerosis and arteriosclerotic dementia.

PRESENILE PHYCHOSES

This leads us to cerebral arteriosclerotic psychoses as such. But before discussing them and the senile dementias proper, I should say a few words about the so-called *presenile psychoses*, which as regards age lie between the involutional psychoses and the old-age group of psychoses.

There are several histological varieties of these presenile psychoses but the best known are Alzheimer's and Pick's diseases. I shall only mention them in passing, because vascular disease does not seem to play an important role in these conditions, although Alzheimer thought that Pick's disease probably had an arteriosclerotic aetiology.

Pick's disease is based on a selective atrophy of parts of the brain (mainly frontal or temporal) due to death of ganglion cells. In Alzheimer's disease the cerebral atrophy is more generalized. Its histology, with its abundance of senile plaques and fibrillary tangles, places it much nearer to the senile psychoses proper. Both diseases cause progressive gross dementia, usually occur in the presenium, but may make their appearance much earlier.

ARTERIOSCLEROTIC PSYCHOSES

As regards *Cerebral Arteriosclerosis* the old division into arteriosclerosis of the large vessels, causing neurological syndromes, and the generalized arteriosclerosis of the smaller cortical vessels, causing psychiatric disease, is still a useful one. It must not be forgotten that cerebral arteriosclerosis can remain symptomless for a long time.

It stands to reason that neurological disease caused by haemorrhage or thrombosis also has its psychiatric aspect, especially in the case of aphasia, but its discussion would lead us too far.

As regards the general cerebral arteriosclerosis one can distinguish between a mild (beginning ?) form causing a neurasthenic syndrome (mentioned above) and a more intense affection of the cortex causing arteriosclerotic dementia.

The prognosis of the mild form need not be too bad, at least not for quite a time; it shows moreover a marked tendency to remission. We must have all treated cases of this kind where with wise handling life remained tolerably normal and active for many years. It would of course be unwise not to take certain precautionary measures but it would be equally unwise to prescribe complete rest or idleness in these cases; in fact one should advise, if possible, continuation at a slower *tempo* of the patient's original work or activities with a mental interest such as creative hobbies. What should be avoided are efforts to live above one's reserve and vascular age and above all undertaking new responsibilities.

The severe form of progressive arteriosclerotic dementia is of course another matter. Its prognosis is poor, its treatment symptomatic.

SENILE PSYCHOSES

It is often impossible to distinguish these cases from *senile dementia;* in fact in post-mortems one usually finds combinations of cerebral arteriosclerotic disease and senile changes. Cases of typical clinical presbyophrenia have been described which later histological examination proved to be chiefly arteriosclerotic and the opposite also holds good.

That does of course not mean that there are no senile dementias without or with very little arteriosclerotic changes. One must not forget that many mild senile dementias have gradually developed *via* the physiological senile mental changes of loss of mental elasticity, of fixation on the past and lack of interest in the present and in consequence of fixation amnesia. Such patients, if one can call them that, may have soft arteries and become centenarians.

Recapitulating, one can therefore say that cerebral vascular disease can play some part in some of the involutional psychoses, little or none in the presenile psychoses and a deciding role in arteriosclerotic psychoses and dementia, and that it probably is also concerned in many cases that have been diagnosed as senile dementia during life.

How far then does cerebral vascular disease play a part in the process of aging?

There is a great deal of truth in the saying: '*On n'a que l'âge de ses artères*,' but that does not mean that the condition of one's blood-vessels is the deciding factor in aging, although it may be a deciding factor in growing old prematurely. After all, we also grow old without arteriosclerosis. It will probably remain a mystery why life's parabola must come to an end in any case, and what causes its curve to go downhill so much later in some people than in others. I suspect that, if we could preserve the harmonious functioning of all our endocrinal glands, life would continue for ever, which is just another way of expressing an insoluble riddle.

One thing is certain, the arteriosclerotic does not make old bones. Atheroma is not a disease of old age, it is a disease of middle and early old age. It would be nice if we could prevent or cure it, we might prolong the life of our patients. But unfortunately it is a sign of old age of the arterial system itself which has come to the end of its span of life and it is to a marked degree hereditary. Again unfortunately, we cannot choose our ancestors, otherwise we might all reach our century, for very old people are almost invariably descended from long-living parents.

But if we cannot help our patient by preventing early arteriosclerosis or by choosing his ancestors for him, we can give him good advice: To remain functioning and not to retire into inactivity. Applied to his mind that means to continue with mental work as long as possible, preferably in his own sphere and otherwise in creative hobbies, which naturally will be different in people of different educational levels, tastes or talents but which should be mental all the same, keeping his mind interested and alive and therefore keeping it young.

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VAN DIE REDAKSIE

SINDROME BY VITAMIEN B12-GEBREK

Dit is noodsaaklik vir die behoorlike rypword van die rooi bloedselle dat daar 'n voldoende konsentrasie van vitamien B12 in die selle is. Hierdie vitamien kom hoofsaaklik in dierlike proteïene voor, en dit is slegs by 'n paar plantaardige voedselstowwe, soos grondboontjies en seewier, afgesonder. Ons liggame trek hierdie stof uit die voedsel wat ons eet en dit word uit die maagdermkanaal in die weefsels opgeneem indien 'n bloedvormende faktor (intrinsieke faktor) deur die maagslymvlies afgeskei word. As hierdie intrinsieke faktor ontbreek, ontwikkel daar megaloblastiese veranderings in die beenmurg en sirkulerende bloed. Die vol-ontwikkelde beeld van so 'n siekte word by Addisoniese kwaadaardige bloedarmoede gesien; hier word die intrinsieke faktor nie afgeskei nie en kom 'n histamien-vaste gebrek aan soutsuur in die maag voor weens verskrompeling van die maagslymvlies. Indien die tipiese kliniese verskynsels teenwoordig is tesame met megaloblastose van die beenmurg, is die diagnose maklik, en 'n gunstige reaksie op behandeling met vitamien B12 kan verwag word.

Dit blyk egter dat 'n gebrek aan vitamien B12 (sianokobalamien), wat goed reageer op behandeling met vitamien B12, ook kan voorkom waar daar *geen* bloedarmoede is nie. 'n Bekende voorbeeld hiervan is die subakute gekombineerde ontaarding van die rugmurg waarby die senuweeletsels oor die jare vererger, egter met normale vorming van rooi bloedliggaampies. Ook kom 'n vitamien B12-tekort voor by pasiënte wat vry suur in die maag afskei en geen tekort aan intrinsieke faktor het nie.

Die enigste betroubare metode om die vitamien B12-tekort direk te bereken is om die hoeveelheid beskikbaar in die liggaam te bepaal. Dit is moontlik gemaak deur die gebruik van die metodes van mikrobiologiese ontleding van die gehalte in die bloedserum.[1-3] Daar is nog 'n ander toets wat indirekte inligting verskaf van die (liggaam se) vermoë om vitamien B12 te absorbeer, nl. Schilling[4] se toets vir die opname van radioaktiewe vitamien B12, waar die urienuitskeiding gemeet word ná mondelikse toediening van die gemerkte kobalt. Gebrekkige absorpsie word aangetoon deur 'n lae gehalte in die urien.

Deur middel van hierdie metodes kan omvangryker navorsing gedoen word op die rol wat sianokobalamien by bloedarmoede en ander siekteverskynsels speel. Dit word algemeen aangeneem dat die normale serumhoogte by 'n volwasse mens tussen 150 en 900 $\mu\mu$g. per ml. is, met 'n gemiddelde hoeveelheid van 390 $\mu\mu$g. per ml. By kwaadaardige bloedarmoede is die gehalte 10-100—gemiddeld 50 $\mu\mu$g. per ml. Dit is moontlik dat die liggaam 'n reserwe

EDITORIAL

SYNDROMES IN VITAMIN B12 DEFICIENCY

An adequate concentration of vitamin B12 in the tissues is necessary for proper maturation of red blood-cells. It is contained mainly in animal proteins, and it has been identified in only a few vegetable foods, such as peanuts and seaweed. The substance is obtained from the food we eat, and it is absorbed into the tissues from the gastro-intestinal tract when a haemopoietic factor (intrinsic factor) is secreted by the mucosa of the stomach. When intrinsic factor is absent, megaloblastic changes will develop in the bone-marrow and circulating blood. The full-blown picture of such a condition is seen in Addisonian pernicious anaemia, in which there is failure of secretion of intrinsic factor and histamin-fast achlorhydria due to atrophy of the gastric mucous membrane. Given typical clinical appearances together with megaloblastosis of the bone-marrow, the diagnosis presents no difficulty and a successful response to treatment by vitamin B12 may be expected.

It has, however, become apparent that vitamin B12 (cyanocobalamin) deficiency, responding well to vitamin-B12 treatment, may occur in the absence of signs of anaemia. A well-known example of this is subacute combined degeneration of the cord, in which neurological defects may progress over a period of years with normal erythropoiesis; and there may be deficiency of the substance in some patients who secrete free acid in the stomach and have no lack of intrinsic factor.

The only reliable method of directly assessing vitamin-B12 deficiency is by measuring the amount available in the body and this was made possible by the introduction of methods of microbiological assay of the level in the blood serum.[1-3] Another test giving indirect information concerning capacity to absorb vitamin B12 is the Schilling[4] modification of absorption of radio-active vitamin B12, whereby the urinary excretion is measured after the oral administration of the labelled cobalt. Deficient utilization is revealed by a low level in the urine.

These techniques have widened the field of research in the part played by cyanocobalamin in the anaemias and other disease conditions. It is generally accepted that the normal adult serum level ranges from 150 to 900 $\mu\mu$g. per ml., with an average of 390 $\mu\mu$g. per ml. In pernicious anaemia the

van 2,000 μg. of meer het van hierdie stof, en dat die gebrek, waar dit voorkom, reeds voorafgegaan is deur 'n lang tydperk van verval. Maande of selfs jare kan verbygaan voordat die kritieke bodempunt bereik word. Pitney en Beard[5] het reekse-bepalings uitgevoer op 'n pasiënt wie se hele maag omrede kanker uitgesny was. Hulle het bevind dat die serumhoogte geleidelik gedaal het en dat dit eers na 8 maande die hoeveelheid van 110 μμg. per ml. bereik het.

Megaloblastiese bloedarmoede kan by 'n verskeidenheid siektes voorkom in gematige en tropiese klimate; idiopatiese steatorrhea en lusse en vernouings van die dunderm waarin die inhoud nie voortbeweeg nie, is voorbeelde hiervan. Mollin en Ross[6] het baanbrekerswerk gedoen op die hoeveelheidsbepaling van vitamien B12 in menslike serum, en in die loop van hulle werk het hulle by meer as 'n derde van die gevalle van steatorrhea lae gehaltes gevind binne skommelingsperke wat by kwaadaardige bloedarmoede verwag kan word. Dit was aangetoon dat pasiënte met subakute gekombineerde ontaarding, sonder bloedarmoede, die laagste gehaltes gehad het. Meynell *et al.*[7] het soortgelyke bevindings by steatorrhea en ook by streek-ileitis beskrywe, maar by verswerende colitis was daar geen vitamien B12-gebrek nie. Adams[8] het onlangs verslag gedoen oor 3 pasiënte wat bloot oor seer tonge gekla het. Daar was so te sê geen bloedarmoede nie, maar die B12-gehaltes was binne die perke wat by kwaadaardige bloedarmoede gevind word, en die pasiënte het besonder goed op behandeling met vitamien B12 gereageer.

In Suid-Afrika slaan die voorkomssyfer van kwaadaardige bloedarmoede by blankes ooreen met dié wat in Europa en Amerika aangetref word. Dit is blykbaar seldsaam by naturelle en Indiërs en dusver is slegs 3 gevalle gerapporteer. Adams[9] beskryf die geval van 'n manlike Zoeloe met makrosiet-anemie, megaloblastiese beenmurg, maagverskrompeling, en 'n serumgehalte aan B12 wat by kwaadaardige bloedarmoede verwag kan word.

Die meeste gevalle van megaloblastiese anemie by die Bantoe- en die Indiërbevolking staan in verband met swangerskap en die kraamtydperk, en die kondisie word goed beskryf in ons literatuur.[10–15] Die patogenese van die siekte word nog nie goed verstaan nie. Die meeste pasiënte skei vry suur af en reageer goed op behandeling met foliensuur, maar 'n paar herstel ná behandeling met groot dosisse vitamien B12. Adams[16] het gevind dat die serumgehalte aan vitamien B12 by die meeste gevalle binne normale perke is en verklaar dat hy ‚nog geen gevalle (gesien het) wat nie op foliensuur gereageer het by die afwesigheid van besmetting nie'. Aangesien dit so moeilik is om te bepaal wat dan juis die faktor is waaraan die liggaam gebrek ıy, blyk dit dat 'n kombinasie van foliensuur en vitamien B12 die verkieslikste behandeling is. Adno[14] het omtrent 7 gevalle van hierdie gebreksindroom by welgevoede, blanke swanger vroue beskryf.

Foy *et al.*[17] klassifiseer die gevalle van nie-kwaadaardige anemie wat hulle in Oos-Afrika by naturelle beskryf in 2 groepe. Die eerste groep reageer op behandeling met penisillien òf op klein dosisse vitamien B12, en die serumgehaltes aan B12 is baie laag. Die tweede groep reageer alleenlik op mondelikse foliensuur en die B12-gehaltes is normaal. Dit word gemeen dat, by dié gevalle wat op penisillien reageer, die antibiotiese middel sekere organismes in die derm vernietig wat met die liggaam kompeteer om vitamien B12. Moontlik staan hierdie kondisie in verband met

level is 10-100, with an average of 50 μμg. per ml. It is possible that the body has a reserve of up to 2,000 μg. or more of the substance and that where deficiency occurs it follows a slow decline, and months or years may elapse before the critical minimum level is reached. Pitney and Beard[5] carried out serial estimations in a patient who had had a total gastrectomy for carcinoma and found that the serum level fell gradually and that only after 8 months had it dropped to 110 μμg. per ml.

Megaloblastic anaemia may occur in a variety of conditions in temperate and tropical climates; idiopathic steatorrhoea and stagnant loops and strictures of the small bowel are examples. Mollin and Ross,[6] in their pioneer work on the assay of vitamin B12 in human serum, found low values in more than one-third of cases of steatorrhoea, some in the range expected in pernicious anaemia. Patients with subacute combined degeneration but no anaemia were shown to have the lowest values. Meynell *et al.*[7] described similar findings in steatorrhoea and also in regional ileitis, but there was no vitamin-B12 deficiency in ulcerative colitis. Adams[8] has recently described 3 patients complaining merely of sore tongue. There was no appreciable anaemia, but B12 levels were in the range noted in pernicious anaemia and response to treatment with vitamin B12 was excellent.

In South Africa the incidence of pernicious anaemia in Europeans is similar to that found in Europe and America. It is apparently uncommon in the African and Indian populations and only 3 such cases have been reported. Adams[9] describes the case of a male Zulu who had a macrocytic anaemia, megaloblastic bone-marrow, gastric atrophy, and a serum-B12 level in the range to be expected in pernicious anaemia.

The majority of cases of megaloblastic anaemia in Bantu and Indian patients are associated with pregnancy and the puerperium, and the condition is well documented in the South African literature.[10–15] The pathogenesis of the condition is as yet uncertain. The majority secrete free acid and respond to treatment by folic acid, but a few recover after therapy with large doses of vitamin B12. Adams[16] finds the serum vitamin-B12 level to be within normal range in the majority of cases and states that he has 'not encountered failure to react to folic acid in the absence of infection'. In view of the difficulty of deciding which of the factors is deficient, the treatment of choice in this type of megaloblastic anaemia would appear to be a combination of folic acid and vitamin B12. Adno[14] has described some 7 cases in well-fed European women during the antenatal period.

The non-pernicious megaloblastic anaemias of Africans described in East Africa by Foy *et al.*[17] are classified by them into 2 categories. The first responds to treatment either by penicillin or small doses of vitamin B12 and the serum-B12 levels are very low. The second group responds only to folic acid by mouth, and vitamin-B12 levels are normal. It is thought that in the penicillin-responsive cases the antibiotic destroys organisms in the gut which compete with the body for vitamin B12. The condition is probably

die dieet, wat arm aan proteïen en ryk aan koolhidraat is. Maar dit is nog nie duidelik of dié dieet te min vitamien B12 en foliensuur bevat nie, òf of dit die ontwikkeling van bakteriese flora aanmoedig wat die behoorlike sintese en absorpsie van die vitamiene teenwerk nie.

Dit is reeds bewys dat 'n goeie persentasie van bejaarde pasiënte met 'n soutsuur-tekort (maar geen anemie nie) vitamien B12 swak absorbeer.[4] Pedersen *et al.*[18] het tydens soortgelyke studies in Denemarke die beenmurg van pasiënte met ligte hipochromiese anemie en achlorhydria bestudeer en gevind dat omtrent 15 persent 'n gedeeltelike of beginnende megaloblastose getoon het. Hulle stel voor dat ligte vitamien B12-gebrek té selde uitgeken word omdat die megaloblastiese neiging deur 'n ystertekort (dimorfies) gemasker kan word. As daar grade van vitamien B12-gebrek bestaan, soos hulle voorstel, wat vae simptome van agteruitgang toon soos seer tong, swakheid en geestesversteurings, is daar in sommige gevalle regverdiging vir geneeshere se aanspraak daarop dat inspuitings van vitamien B12, veral by bejaardes, 'n ‚opknappende' uitwerking het.

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9. Adams, E. R. (1957): S. Afr. T. Geneesk., **31,** 633.
10. Cohen, L. (1953): *Ibid.*, **27,** 627.
11. Adams, E. B. en Wilmot, A. J. (1953): *Ibid.*, **27,** 1028.
12. *Idem.* (1956): Brit. Med. J., **2,** 398.
13. Patz., I. M. (1957): S. Afr. T. Geneesk., **31,** 384.
14. Adno., J. (1957): *Ibid.*, **31,** 10.
15. Nel, R. W. A. (1957): *Ibid.*, **31,** 192.
16. Adams, E. B. (1957): *Ibid.*, **31,** 324.
17. Foy, H., Kondi, A. en Manson-Bahr, P. E. C. (1955): Lancet, **2,** 693.
18. Pedersen, J., Lund, J., Ohlsen, A. S. en Kristensen, H. P. O. (1957): *Ibid.*, **1,** 448.

related to the diet, which is poor in protein and rich in carbohydrates. But it is not as yet clear whether the diet is deficient in vitamin B12 and folic acid, or encourages the growth of bacterial flora inimical to proper synthesis or absorption of the vitamins.

It has been shown that a fair proportion of elderly patients with achlorhydria but no anaemia absorb vitamin B12 poorly.[4] In parallel studies in Denmark, Pedersen *et al.*[18] examined the bone marrow of patients with mild hypochromic anaemia and achlorhydria and found that some 15% had a partial or incipient megaloblastosis. They suggest that mild vitamin-B12 deficiency is too seldom diagnosed because the megaloblastic tendency may be masked by iron deficiency anaemia (dimorphous). If, as they suggest, there are degrees of vitamin-B12 deficiency producing vague symptoms of ill-health, such as sore tongue, weakness and psychological disturbances, there would be some justification, in occasional cases, for the insistence by practitioners that injection of vitamin B12 has a 'tonic' effect, particularly in elderly patients.

1. Hutner, S. H., Provasoli, L., Stokstad, E. L. R., Hoffman, O. E., Belt, M., Franklin, A. L. and Jukes, T. H. (1949): Proc. Soc. Exp. Biol., **70,** 118.
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ILLNESS AMONG NATAL INDIANS: A SURVEY OF HOSPITAL ADMISSIONS

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It has been observed that the pattern of disease among Asiatics in Durban resembles that in Europeans more closely than that in the Bantu population. There are, however, certain exceptions. The object of this survey has been to analyse the conditions for which Asiatics (Indians) are admitted to hospital, to provide impressions on the relative frequency of certain diseases and to illustrate those features of disease that are peculiar to Indians.

Unfortunately, in many instances, it is not possible to supply more than personal impressions of the relative frequency of diseases among the racial groups in Durban. Statistical comparison fails because of the lack of local data regarding morbidity in these races and because of different conditions pertaining to their admission to hospital. The publications of the General Register Office[1] serve as a guide to disease incidence among the population of England and Wales. Figures regarding Bantu patients at this hospital have been used for comparison in some instances.

Material

This survey is based on the clinical records of 10,000 patients admitted consecutively to the Indian wards of

King Edward VIII Hospital over a 16-month period ending in April 1956. It is estimated that the Indian wards of this hospital handle about 65% of hospital admissions in an area whose Asiatic population is approximately 250,000. It is thus likely that the patients in this series represent illness among some 160,000 individuals.

Since the wealthier classes obtain private medical attention and are usually admitted to other hospitals, there exists a slight bias in favour of the poorer classes among the patients at this hospital. Infectious diseases and some cases of tuberculosis are treated in separate units, so that the admissions for these conditions are lower than the true incidence.

Approximately 1·3% of the hospital records were missing or not available for analysis during the period occupied by this study. It is reasonably certain that there has been no selective loss in this respect.

Table I shows the conditions most commonly encountered, in order of frequency.

TABLE I. COMMONEST REASONS FOR ADMISSION IN ORDER OF FREQUENCY

	Percentage
Obstetric (including all complications of pregnancy, BBA and false labour)	21·0
Injuries of all kinds	11·4
Abortions	1·5 to 2·0
Superficial infections and abscesses	,,
Diabetes mellitus	,,
Gastro-enteritis	,,
Appendicitis	,,
Rheumatic fever and sequelae	1·0 to 1·5
Bronchopneumonia	,,
Tuberculosis of lungs or pleura	,,
Malnutrition	,,
Peptic ulcer	,,
Cerebral vascular accidents	,,
Amoebiasis (dysentery or liver abscess)	0·5 to 1·0
Hypertension and hypertensive heart failure	,,
Iron-deficiency anaemia (including hookworm)	,,
Burns	,,
Nephritis (all types)	,,
Bronchial asthma	,,
Pelvic infections	,,
Poisoning	,,
Worm Infestations (excluding hookworm anaemia)	,,
Pyelitis	,,
Haemorrhoids	,,
Undiagnosed on discharge	3·6

GASTRO-INTESTINAL CONDITIONS

Gastro-enteritis, bacillary dysentery and amoebic infections account for over 3% of hospital admissions. These are somewhat less common than among the Bantu, but a more striking difference is the greater resistance which Indians seem to possess against these infections. Indian infants with gastro-enteritis do not often show the severe dehydration, toxaemia and collapse which is common among Africans, and their mortality rate is lower.

Similarly among adult Indians severe fulminating amoebic dysentery has not been encountered. All forms of amoebic infection are less dangerous. In this respect the disease pattern resembles that of amoebiasis in the European population. Amoebic liver abscess is less common among Indians than Africans and is better tolerated by the host. Rupture of an amoebic abscess is a rare event.

Worm infestations are common. Ascariasis and trichuriasis are almost universal. Hookworm is less common but, in its relationship to iron deficiency anaemia, is responsible for greater morbidity than any other parasite among the Indians. Not one case of tapeworm infestation occurred among 10,000 patients. Consumption of pork is not prohibited by the Hindus, who constitute the majority of patients at this hospital. It is likely that the method of preparation of food is responsible for the rarity of tapeworms among the Indian population.

Ova of *Schistosoma mansoni* are found in the stools as commonly as ova of *S. haematobium* in the urine. The extent to which intestinal bilharzia is productive of symptoms is difficult to evaluate. Several cases of persistent abdominal pain occurred in which the only abnormal finding was that of bilharzia ova in the stools. Some cases of bilharzial appendicitis have been proved histologically.

Peptic ulcers are common among Indians but rare in the Bantu. In Indians the incidence with regard to sex, age and situation is similar to that found among Europeans.

A striking difference in racial incidence occurs in gall-bladder disease. Stocks reports that the monthly prevalence rate of these conditions in England and Wales was 121 per 100,000 population.[1] The local European population seems equally susceptible. In this series of Indian patients over a 16-month period there occurred only 7 cases with disease of the gall-bladder. Of these, 5 had gall-stones and 2 showed clinical and radiological features of chronic cholecystitis. Three of the patients were diabetics. The extreme rarity of these conditions is not easily explained. Factors which are commonly associated with cholecystitis are prevalent among Indians. Obesity and diabetes are common and fecundity is frequently phenomenal.

CARDIOVASCULAR DISEASE

Rheumatic fever and rheumatic heart disease are exceptionally common among the Indians. Of all conditions treated in medical wards they are only slightly less frequent than diabetes and gastro-enteritis. Rheumatic carditis and its sequelae account for more deaths in the 10-30 year age-groups than all other medical conditions combined. Acute rheumatic fever is rarely seen among Bantu patients, though the sequelae are more commonly observed. Rheumatic fever and carditis appear in a more florid form among the Indian children, deaths from this cause are much commoner, and valvular lesions more extensive than in European or Bantu children. In spite of the high incidence of rheumatic heart disease we have been able to recommend mitral valvotomy in very few patients. Relapses are so frequent and multiple valve lesions develop so rapidly, that cases suitable for surgery are rarely encountered.

Hypertensive heart disease, coronary thrombosis and cor pulmonale are relatively common and follow the pattern observed among Europeans. Coronary thrombosis is much more frequent than among the Bantu.

In this series were also several patients with unexplained cardiac failure. Most of these conform to the type which has been called nutritional heart disease among the Africans.

Varicose veins were the commonest type of peripheral vascular disease observed, but the incidence is considerably

lower than among Europeans. A study of the sex incidence in this series reveals an unusual distribution. Of all patients who were admitted to hospital on account of varicose veins, 4/5ths were males. This is all the more surprising in view of the high degree of parity of Indian women. It appears that pregnancy is not a significant factor in the genesis of varicose veins in Indians.

ANAEMIAS AND BLOOD DISORDERS

Severe anaemias are remarkably common among the Indian population, and the nature of these anaemias differs from that found among the European and Bantu. Among 10,000 admissions there were 128 patients whose haemoglobin

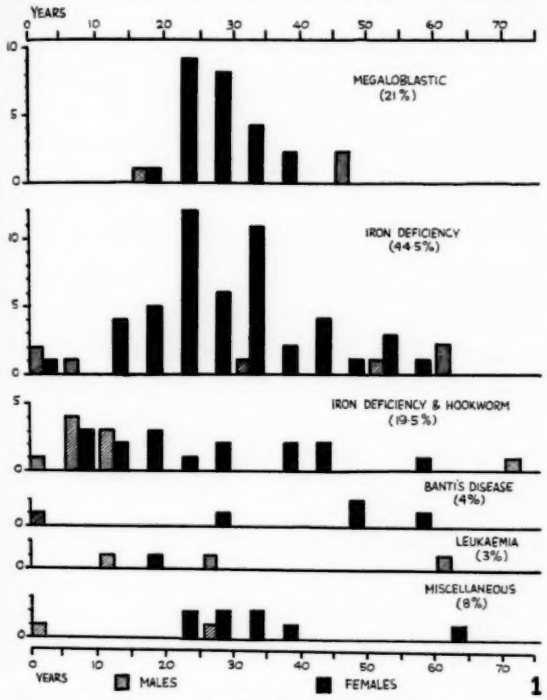

Fig. 1. Severe anaemias: distribution according to type, age and sex.

levels were below 7·4 g.% (50%). Patients with anaemia due to frank haemorrhage and surgical or gynaecological causes are excluded from this group. The distribution of the cases according to age, sex and type is shown in Fig. 1. The arbitrary level of 7·4 g.% haemoglobin has been chosen for the sake of simplicity in classification. Less severe anaemias frequently receive out-patient treatment.

Iron-deficiency anaemia accounts for about 2/3rds of all severe anaemias. In this respect the Indians differ markedly from the Africans, in whom severe iron-deficiency is rare. In about 1/3rd of Indian patients with iron-deficiency anaemia, ova of hookworm have been found in the stools after one or two examinations. The role of hookworm in the production of anaemia is not settled. Some authors have cast doubt on any relationship between the two conditions. The evidence of this group of patients distinctly incriminates the hookworm. Occult blood has been present in the stools of the majority of our patients with severe anaemia and hookworm infestation. Eosinophilia was present in many of these patients.

In about 2/3rds of the iron-deficiency group no cause is directly obvious. The age and sex distribution (Fig. 1) emphasizes the importance of the demands made by menstruation and pregnancy. It is also significant that among males iron-deficiency anaemia occurs almost exclusively below the age of 15 years. It is reasonable to suppose that, because of a metabolic or dietary deficiency, the iron balance of many Asiatics is in a precarious state. Any increased demand, such as those of growth, menstruation, pregnancy or hookworm infestation, is sufficient to make the defect clinically manifest. The rarity of iron deficiency among the Africans indicates some difference in the iron metabolism of these two races.

The lowest haemoglobin level observed among these patients with iron-deficiency anaemia was 1·6 g.% (11%).

Megaloblastic anaemias of pregnancy and the puerperium are well recognized among Indian and Bantu patients at this hospital.[2, 3] In the present series there were 27 cases of megaloblastic anaemia (Fig. 1). Of these, 23 occurred in association with pregnancy and the puerperium; 2 patients presented in the last trimester of pregnancy, 4 were at term, and the remaining 17 presented 1-9 months after delivery.

There were 4 cases of megaloblastic anaemia unrelated to childbirth. One was a man with carcinoma of the anus who died before investigations were complete. The remaining 3 patients, 2 men and 1 woman, belong to a type not previously described among Durban Indians. The feature common to the 3 patients was that, for religious or personal reasons, none of them consumed any meat, fish or animal products. The following case illustrates the features of this type:

A man of 20 years was admitted severely ill in congestive cardiac failure with a haemoglobin of 2·7 g.%. His religion forbade the consumption of any animal foods. The diet had been adequate in other respects. In addition to signs of congestive cardiac failure with a dilated heart and widespread haemic murmurs he had mild jaundice, splenomegaly and numerous retinal haemorrhages with a few soft exudates. Serum bilirubin was 4 mg.%. Numerous megaloblasts were present in the peripheral blood and the diagnosis was confirmed by bone-marrow biopsy. Free HCl was present in the gastric juice. In view of the presumed dietary deficiency of extrinsic factor he was treated with vitamin B12 in doses of 100 μg. daily by injection. There was a rapid reticulocyte response, which reached a maximum of 53% on the 7th day, followed by complete recovery.

The other two cases of this vegetarian type of megaloblastic anaemia were a man of 46 and a woman of 40. The latter was treated with vitamin B12 by mouth in doses of 150 μg. daily. This produced a reticulocyte response of 22% on the 14th day and eventual complete recovery. None of these patients showed evidence of neurological disease.

It is possible that megaloblastic anaemias may be divided into two types depending on the serum-B12 levels. Those with normal levels, such as the anaemia of pregnancy, usually respond to treatment with folic acid. In pernicious anaemia the serum-B12 levels are subnormal. Megaloblastic anaemias in vegetarians have been described by several authors.[4, 5, 6] This anaemia, which apparently represents a pure dietary deficiency of extrinsic factor, resembles pernicious anaemia in respect of low serum-B12 levels, response to vitamin B12 and possible development of neurological complications. Our 3 cases are presumably of this type. A further feature

in these cases was the presence of jaundice, which appears to be more pronounced in cases of B12 deficiency than in the megaloblastic anaemia of pregnancy.

Retinal haemorrhages have been observed in many of our cases of megaloblastic anaemia, but have not been seen in iron-deficiency anaemia of comparable severity. This sign has received scant mention in the literature and deserves greater emphasis since it has been found to have diagnostic significance. Other evidence of a haemorrhagic tendency, such as vaginal or gingival bleeding, has also been present in some cases of megaloblastic anaemia.

All cases of megaloblastic anaemia associated with pregnancy were treated with folic acid. The response was usually good. Some patients have become deficient in iron while under treatment.

There were 2 deaths from megaloblastic anaemia in this series. Both were severe anaemias presenting in the puerperium with haemoglobin levels of 2·2 g.% (15%) and 1·4 g.% (10%) respectively. These were the only patients in this group who received blood transfusions. No inference can be drawn, since these were also the two most severe cases of megaloblastic anaemia of the group. The response of other patients to the appropriate haematinic substance has been so prompt that one may conclude that blood transfusion is only justified when the usual treatment fails.

Very severe degrees of anaemia occurred in both iron-deficient and megaloblastic groups. Patients with the former disease are generally much less disabled than patients with megaloblastic anaemia of comparable severity. Cardiac failure occurs more readily in megaloblastic anaemia. Iron-deficient patients are often remarkably well despite alarming haemoglobin levels.

Unexplained *splenomegaly* has been observed, most frequently as an incidental finding in female patients. This is often associated with varying degrees of iron-deficiency anaemia. Where the anaemia is severe and associated with gastro-intestinal bleeding and leucopenia the presumptive diagnosis has been Banti's syndrome.

NEUROLOGICAL DISORDERS

The incidence and nature of most neurological disorders in Indians runs parallel to those found among the European population. Cerebral vascular accidents, meningitis, epilepsy, peripheral neuritis and tumours occur in that order of frequency.

There remains a group of conditions in which the diagnosis is written with a question mark. This usually implies that the patient has had undoubted organic neurological disease but that it has been difficult to relate it to any definite clinical entity. Fortunately most of these conditions are self-limiting and the patients usually recover with little specific treatment. These obscure nervous disorders may be divided into two groups.

The first and larger group probably represents *virus infections* of the nervous system. In this category are patients with atypical encephalitis, meningitis, myelitis or unexplained cranial- or peripheral-nerve palsies. In these cases the cerebrospinal fluid has been normal or has shown a variety of changes of which the commonest is slight pleocytosis and increased protein content with no alteration in other chemical constituents.

In the second group of obscure conditions blame is usually laid on malnutrition. Various nutritional neuropathies have been described, but there seems to be no consistency in the neurological picture which can be produced by malnutrition. Few of our cases conform to the types described but, in the presence of undoubted evidence of malnutrition and response to treatment, one is forced to the conclusion that the neurological disorder is due to this cause. The following case is an example:

A 43-year-old woman had increasing difficulty with walking for 1 year. On admission she was unable to walk without assistance and had signs of a spastic paraplegia with exaggerated tendon reflexes and bilateral ankle clonus. Some intellectual impairment made examination of sensation difficult but it appeared that both superficial and deep sensation in the legs were deficient. She had signs of mild pellagra and an iron-deficiency anaemia with a haemoglobin level of 5·1 g.%. Investigations, including that of the cerebrospinal fluid, showed no further abnormality. Within 4 weeks she had recovered fully. Treatment consisted of normal hospital diet, vitamin supplements and iron.

Severe iron-deficiency anaemia was a feature of several other cases of nutritional neuropathy, but since the former is common in this race it may not have any direct relationship to the latter.

There are other neurological conditions whose incidence among the Indians is unexpectedly high. Rheumatic chorea is common and its frequency parallels the high incidence of rheumatic fever among Indian children. Of intracranial space-occupying lesions, tuberculomata, either single or multiple, are nearly as common as neoplastic growths. In these cases diagnosis is usually based on the discovery of other tuberculous lesions and on response to treatment. In one fatal case multiple cerebral tuberculomata were found *post mortem* to be associated with tuberculous endometritis.

Muscular dystrophies appear to be unusually prevalent in Indians. Among 10,000 admissions there have been 8 cases of muscular dystrophy, representing 5 unrelated families. Within the families the dystrophies breed true in type. Outside the familial relationship there was wide variation in the type of dystrophy encountered.

Familial cerebellar ataxia of the Holmes type was seen in 2 middle-aged brothers.

Disseminated sclerosis has not appeared in our Indian patients.

MALNUTRITION

Malnutrition is exceptionally common and is directly or indirectly responsible for a large number of deaths among Indian infants. Kwashiorkor is seen frequently, often complicated by gastro-enteritis or bronchopneumonia. Among adults, pellagra and multiple deficiencies are common.

Less common, but worthy of mention in that an underlying cause of malnutrition may be overlooked, is the malabsorption syndrome due to abdominal tuberculosis. This has been seen in both adults and children. That medical treatment alone may be inadequate was shown in one case in which the pathology was revealed at autopsy.

A woman aged 27 years was admitted to hospital on 3 occasions. The first time she presented with diarrhoea of 1½ years duration and signs of pellagra. She had previously received treatment for tuberculous cervical adenitis. A barium meal revealed the disordered pattern and intestinal hurry which is associated with

malabsorption. On treatment with streptomycin and isoniazid there was marked improvement. On her second admission the main complaint was colicky abdominal pain, but the clinical picture and response were similar. Treatment of the patient was continued as an out-patient. She returned 9 months later when she was grossly emaciated with oedema of the ankles and sacrum. The main complaint was muscular cramps and she displayed signs of tetany. She had pellagra, a normocytic normochromic anaemia (Hb. 8·8 g.%), hypoproteinaemia (albumen 1·1 g.% globulin 3·5%) and the serum calcium was 5·8 mg.%. Despite treatment she died a week later. At autopsy, apart from a terminal pneumonia, the only further abnormality was an indurated fibrotic stricture involving the ileo-caecal valve and terminal ileum. It seems likely that the malabsorption resulted from the effects of a chronic stricture, the tuberculous process being then inactive.

Several further cases have been seen in which a similar pathological process has been suspected. One middle-aged man, who had been admitted to hospital repeatedly with malnutrition and anaemia over a period of 12 years, has recently shown considerable improvement after resection of a similarly affected portion of bowel. In this case there was, in addition, a blind loop, the result of an ileo-transverse colostomy 3 years previously.

Unfortunately, in these cases of malabsorption due to tuberculosis, the diagnosis is not easily proved and assessment of the need for surgery is equally difficult.

ENDOCRINE DISORDERS

With the exception of diabetes, endocrine diseases are rare. In this series of 10,000 Indian patients there were 11 cases of non-toxic thyroid adenoma and 4 cases of colloid goitre. Thyrotoxicosis is extremely uncommon, no cases having occurred in this series. Myxoedema and hypoparathyroidism occurred in isolated instances, and there were 3 cases of Addison's disease.

Diabetes mellitus, on the other hand, accounted for more admissions to the medical wards than any other single disease. In this series the incidence of diabetics is 19·2 per 1,000 Indian patients. During an equivalant period the incidence among Bantu patients at this hospital was 0·53 per 1,000 admissions. Per unit of population the incidence of diabetes among the Indians appears to be higher than that in England and Wales.[1] Judged by hospital admissions, it is certainly much commoner than among the local European population. Though these data are not strictly comparable, it is obvious that diabetes is exceptionally common among the Indians; and is 30 to 40 times as common as in the local Bantu population. The sex distribution of diabetics follows the usual pattern for other races (Fig. 2). The preponderance of females over males, however, becomes apparent about 10-15 years earlier than it does in a European population.[7,8] Even the men appear to develop diabetes at a somewhat earlier age.

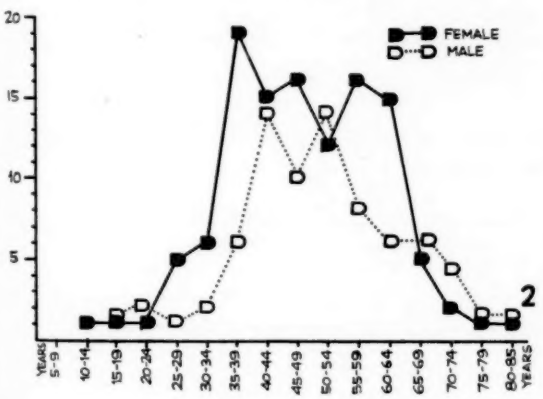

Fig. 2. Age and sex distribution of 192 diabetics.

Besides its frequency and age incidence, diabetes among the Indians exhibits some other unusual features. Ketosis is rare; among the 192 diabetics in this series only 2 were in coma or pre-coma, and less severe ketosis was similarily uncommon. There is also a high degree of insulin resistance among Indian diabetics, the average dose of insulin required being well above the 20-50 units daily quoted by Stocks[1] for English diabetics. Hypoglycaemic reactions are fortunately uncommon. This rarity of ketosis and hypoglycaemic reactions cannot be ascribed to good control of diabetes. Social and economic factors, lack of education, and misunderstanding all contribute towards great difficulty in the control of diabetics by diet and insulin.

In contrast, the other complications of diabetes are extremely common, and these probably reflect more accurately the delay in diagnosis and lack of effective control. Infections and ocular, renal and vascular complications are the commonest reasons for admission of diabetics to hospital. Indeed, the latter two complications are so common and ketosis so rare that experience has shown that coma in an Indian diabetic is more usually the result of renal failure or cerebral vascular accident than ketosis. Diabetic intercapillary glomerulosclerosis has been seen frequently in all stages of development and constitutes one of the gravest complications of diabetes in this race.

Some of our diabetics conform to the J type of diabetics described by Hugh-Jones among Jamaicans.[9,10] This term is applied chiefly to young diabetics who show insulin resistance but do not readily develop ketosis without insulin.

Two factors may be responsible for the unusual pattern and exceptional frequency of diabetes among Indians, one an inherent racial or genetic factor, the other dietary. A more detailed analysis of a further series of Indian diabetics is being made.

ARTHRITIC CONDITIONS

Rheumatic fever is the commonest form of arthritis in this race. Rheumatoid arthritis, gout, and ankylosing spondylitis, all occur less frequently than among Europeans. Excluding rheumatic fever, the commonest form of arthritis is an atypical one which does not conform to the European prototypes. The patient is usually a young adult who presents with an effusion into a large joint, usually the knee. There is, or has been, pain in other joints, but it does not have a flitting character. The joint effusion is clear and sterile on culture. There is no evidence of past or present gonococcal infection. X-rays of the affected joints show no abnormalities apart from occasional slight osteoporosis. The condition usually clears up after 2 or 3 weeks rest and treatment with salicylates. Recurrence is unusual. These cases have been diversely labelled atypical or monarticular

rheumatoid arthritis or palindromic rheumatism, but none of the descriptions can be considered accurate.

MALIGNANT DISEASE

Among 10,000 Indian admissions there were 90 patients with malignant disease. In this group diagnosis was made on pathological or histological grounds, or based on sound radiological evidence. Doubtful cases were excluded. Fig. 3 illustrates the types of malignant disease encountered most

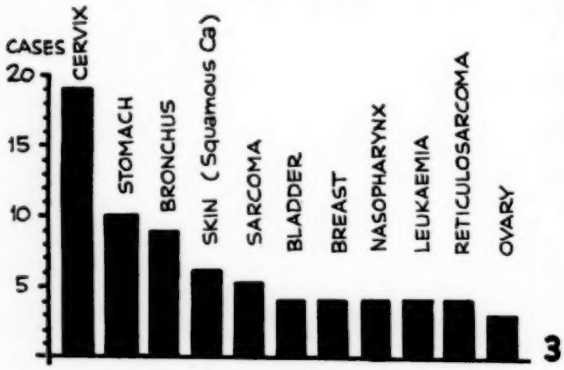

Fig. 3. Commonest types of malignant disease.

frequently among Indians. There were 19 cases of carcinoma of the cervix. Of these, 17 were Hindu and 2 Christian. No cases of cervical carcinoma occurred among Moslems, the only Indian group who regularly practise circumcision. This is in accord with the finding of Wynder[11] that the disease is much commoner among races who do not practise circumcision. Since Hindus predominate in the population there exists a bias in favour of this group.

Carcinoma of the breast (4 cases), colon (2 cases) and rectum (2 cases) have a lower incidence than in the European population. The rarity of breast cancer may be related to early childbearing, large families, and the late weaning which is customary among the Indians.

Primary carcinoma of the liver, common in the local Bantu population, has not been observed in Indians. Of 10 cases of gastric carcinoma all but one occured in males. Among the rare types of malignant disease were 2 cases of sarcoma botryoides in infants.

OBSTETRICS AND GYNAECOLOGY

The first impression gained from a study of Indian maternity records is one of early childbearing and great fecundity. An analysis of the relationship between age and parity, based on the records of 300 consecutive uncomplicated maternity cases gives the following results:

The average age of primiparae was 20·75 years. The average age of women bearing their 5th child was 25·7 years, and that of mothers in labour for the 10th time 34·0 years. Thus the average interval between the births of an Indian mother's first 5 children is 12 months, and between the births of her second 5 children 20 months. Since these figures are culled from hospital maternity records they do not represent the whole population.

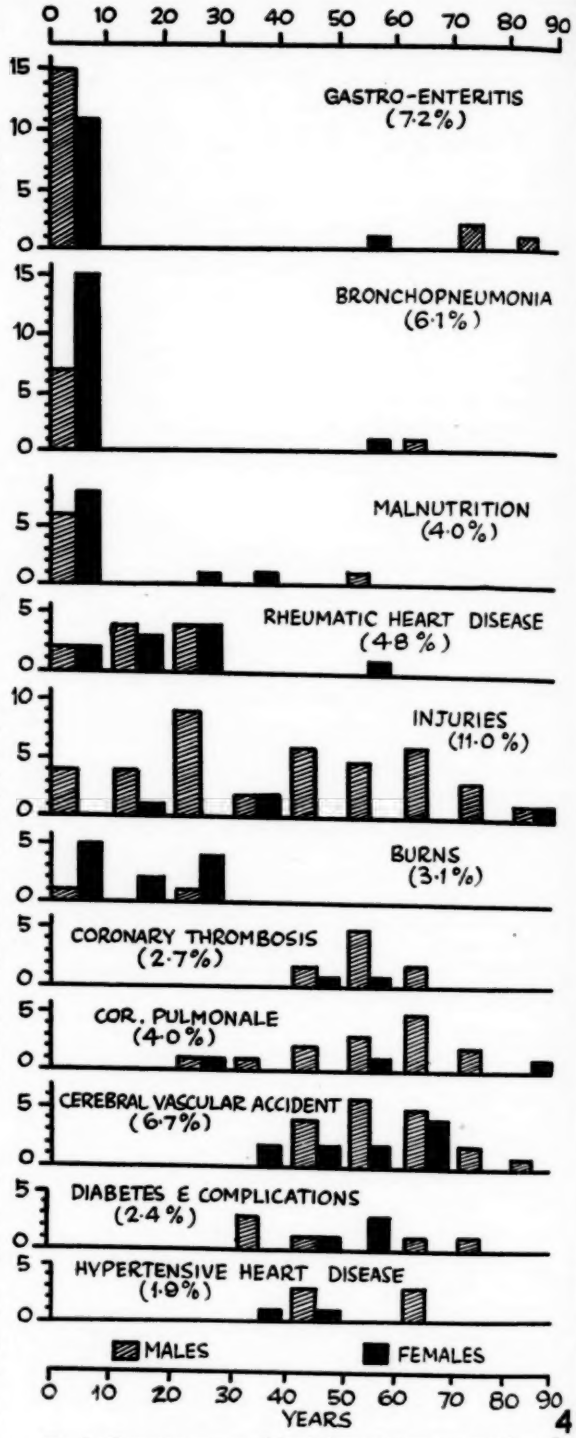

Fig. 4. Common causes of death (percentages expressed as of total deaths—415).

Abortion is the commonest reason for admission to gynaecological wards, and accounts for about 2% of all admissions.

Only 4 cases of fibromyoma of the uterus occured among 10,000 admissions. This rarity may be related to early childbearing and large families.

OTHER DISEASES

Genito-urinary and Respiratory Diseases. Nothing unusual was noted in the incidence or nature of genito-urinary or respiratory disorders among the Indians.

Undiagnosed Cases. Approximately 3·6% of all cases admitted to hospital were discharged without a definite diagnosis having been made. An analysis of these showed that abdominal pain was the main complaint in over 50% of these patients. Most of them were in the 10-30 years age-group, and there was a slight preponderance of females. The stay in hospital was usually short, because the symptoms subsided within a week in most cases. The commonest and frequently the only abnormality found on investigation was evidence of worm infestation, either ascaris or hookworm, or both. Since these parasites are so common in the Indian population the significance of this finding is difficult to assess. In some cases the abdominal pain ceased after anthelminthic treatment. In many others the symptom abated without specific treatment. Undoubted cases of intestinal obstruction due to roundworms have been seen in children. It is reasonable to suppose that less severe symptoms may occur from a similar cause. Other common presenting symptoms in subsequently undiagnosed cases were headache, backache and fever.

DEATHS

It is unfortunate that permission for post-mortem examinations is so rarely forthcoming in this race. Valuable knowledge is repeatedly lost and clinical research is considerably hampered. Some enlightenment is necessary if problems peculiar to this race are to be adequately investigated.

Among 10,000 Indian admissions 415 deaths occurred. The age and sex incidence of some of the commoner causes of death are illustrated in Fig. 4. Over one-quarter of all deaths occurred in the 0-10 years age-group. This high mortality is mainly attributable to gastro-enteritis, bronchopneumonia and malnutrition. Deaths in the next 2 decades reflect the heavy toll taken by rheumatic heart disease and by fatal injuries. The latter account for considerable mortality at all ages, and are almost entirely confined to males. In middle age, coronary thrombosis, hypertension, cerebral vascular disease, and chronic respiratory disorders, are responsible for most deaths.

SUMMARY

Ten thousand consecutive admissions to the Indian (Asiatic) wards of King Edward VIII Hospital, Durban, have been analysed. The conditions which occur with undue frequency or undue rarity, or which show features peculiar to the race, are discussed (Table II).

TABLE II

Unexpectedly common conditions	*Unexpectedly rare conditions*	*Diseases with unusual features*
Rheumatic fever	Cholecystitis	Diabetes mellitus
Rheumatic heart disease	Gall-stones	'Rheumatoid' arthritis
Diabetes mellitus	Thyrotoxicosis	Some anaemias
Iron-deficiency anaemia	Diabetic ketosis	Some neurological disorders
Megaloblastic anaemia	Carcinoma of breast	
Rheumatic chorea	Carcinoma of colon	
Muscular dystrophy	Fibromyoma of uterus	
	Varicose veins	
	Tapeworm infestation	

I wish to thank Dr. S. Disler, Medical Superintendent, for permission to publish. This investigation was suggested by Dr. N. A. Rossiter, to whom I am grateful for interest and advice. My thanks are also due to Miss J. McCrossin, Miss M. van der Merwe and the staff of the records office for their help.

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A REVIEW OF FLUOTHANE *

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The advent of a new drug into the world of anaesthesia is an event of considerable interest, particularly when it is no mere variation on a proprietary theme. Fluothane, is certainly not this, nor indeed does it belong to the group of substituted ethers such as methyl-propyl (Neothyl), vinyl-ethyl (Vinomar), or trifluoro-ethyl-vinyl (Fluomar). It is original in being a halogenated ethane, a chemical group which, as such, has received little attention since John Snow advocated the use of dichlorethane in 1851.[1] In fact, it was while Snow was describing the clinical use of dichlorethane in the final chapter of his book 'On Chloroform and Other Anaesthetics' that he was seized with the illness

* A paper presented at the South African Medical Congress, Durban, September 1957.

which led to his untimely death in 1858 at the age of 40. However, one should perhaps remember that the simplest chlorinated ethane in everyday use is ethyl chloride.

Compared with other well-known anaesthetic agents, Fluothane is reasonably stable. But even an anaesthetist can recognize that the halogen acids might be potential breakdown products of a substance with this chemical structure:

It is known that fluorine is more electro-negative than the other halogens, and therefore one would expect these atoms to be firmly bound to their corresponding carbon atoms. At the same time this strong electro-negative potential tends to unbalance and weaken the linkage to the other carbon atom so that, on theoretical grounds, the most likely consequence of any decomposition would be the liberation of bromine and chlorine. Clinically there has been no evidence of the effects of any decomposition, although it is known to occur in the presence of light with moist air or oxygen. Even in the absence of light, moist air or oxygen allows a rapid reaction to occur between certain metal alloys and Fluothane vapour. This may take the form of a crust of tin bromide or chloride on the tin-foil lining of bottle caps, or actual pitting of aluminium surfaces. This property is of great importance in constructing a vaporizer from which accurately measured concentrations must be delivered; corrosion will take place unless suitable metals are employed throughout, or aluminium and tin surfaces (also any soldered seams) are 'protected' by anodizing, or by coating with nylon or other resistant plastics. Apart from the technical problems involved—and these can be overcome—there is no evidence at present to suggest that decomposition should be regarded any more seriously than it is in other well-known anaesthetic agents.

By far the most important property of Fluothane is its non-flammability in air and non-explosibility in oxygen in any mixture. On these grounds alone the drug merits a thorough assessment to see how far it answers the anaesthetist's criterion of a 'non-flammable ether' in terms of all-round efficiency and safety. The physical, chemical, and pharmacological properties of Fluothane have already been described in some detail.[2,3] Certain aspects undoubtedly need to be clarified, particularly the mechanisms by which the circulatory, respiratory, and sympathetic nervous systems are affected during anaesthesia. However, a discussion on these points would be profitless at this time; the sole object of this paper is to review the clinical aspects of its use.

The evidence is based on rather more than a thousand administrations in which Fluothane has been the sole or principal anaesthetic agent. Although there was no other selection of patients by age, physical fitness, or type of operation, it was not used in the more heroic procedures. Furthermore, it was tested on a number of volunteers to determine the effects of varying concentrations. Administrations were by the open drop technique, from a Boyle-type apparatus with nitrous oxide and oxygen, or from an E.M.O. inhaler[4] which had been specially calibrated and modified for use with Fluothane.

In unpremedicated subjects it was found that 0·5% Fluothane in air had little effect. Prolonged inhalation in this concentration caused no loss of consciousness and little, if any, analgesia, but 1% Fluothane in air regularly produced unconsciousness, and often a sufficient depth of anaesthesia for simple operations. In normal clinical use, slightly higher concentrations were needed to produce muscular relaxation, but it was never necessary to exceed 3%. However, even with this concentration, it was not always possible to provide ideal conditions for upper abdominal surgery. This may well have been the result of depressed respiration limiting the quantity of drug inhaled, a situation comparable to that found during cyclopropane anaesthesia, and one which can be overcome by assisting respiration.

TECHNIQUE OF ANAESTHESIA

Induction

Induction can be carried out with Fluothane alone,[5] or combined with nitrous oxide. In all instances consciousness is lost rapidly, without the accompanying sensation of suffocation so common with most inhalation anaesthetics. Coughing and breath-holding are rare, although occasional periods of apnoea within the first few minutes are not unknown. These do not appear to be associated with laryngeal spasm, for inflation of the lungs may be performed easily. In some cases, but by no means always, the condition is probably caused by a high concentration of Fluothane vapour. Since it may persist for several minutes, inflation is often necessary to avoid cyanosis, particularly if respiration has not already been restarted by applying a painful stimulus to the patient.

An excitement stage is unusual, and can be avoided, as also can the apnoea of induction, by a preliminary injection of thiopentone. For these reasons alone it is helpful to render the patient unconscious with a 'sleep dose' of thiopentone before proceeding with the administration. Repeated swallowing is not uncommon during the early stages of anaesthesia, but can be ignored since it does not presage coughing, breath-holding, or vomiting. At the same time, flushing of the skin, suffusion of the conjunctiva, and dilatation of the superficial vessels, are noticed. These seem to be more marked than under other forms of general anaesthesia and suggest an early effect on the sympathetic system. The observation has no practical significance, but the anaesthetist will appreciate another effect on the autonomic nervous system which causes depression of pharyngeal and laryngeal reflexes. Thus, with early relaxation of the masseter muscles, an oral airway may be inserted without provoking any response at a stage when the patient is still capable of moving. Further, even more vigorous stimulation such as touching the glottis with an endotracheal tube will not initiate spasm. At this stage the cords may come together, or there may be a cough, but normal respiration will be resumed within half a minute.

Maintenance of Anaesthesia

Anaesthesia may be maintained with Fluothane alone or combined with nitrous oxide and oxygen for all types of surgery except, as has been suggested already, where an

extreme degree of relaxation may be required for an upper abdominal operation. In any case, there is probably no justification for attempting to produce such deep anaesthesia by administering the higher concentrations of Fluothane for more than short periods of time. This is because of the effects on the respiratory and circulatory systems which, although apparent early on, undoubtedly vary with the concentration of vapour inhaled. To overcome this difficulty, thoughts immediately turn to the use of muscle relaxants. Of these, small doses only are needed to produce the desired results, but while suxamethonium may be administered as with any other anaesthetic sequence, d-tubocurarine and gallamine deserve special mention. In other instances, particularly in children, where for any reason prolonged anaesthesia with Fluothane is considered undesirable, ether would be the agent of choice. The change-over can be effected smoothly and quickly without risk of coughing or laryngeal spasm. Also, being a respiratory stimulant, the inhalation of ether will counteract any respiratory depression due to Fluothane.

Effect of Fluothane on the Respiratory System

Probably the most significant effect that Fluothane exerts on the body is that on the respiratory system.[6] Even in volunteers who had received no premedication and no preliminary injection of thiopentone, respiration could be reduced to a dangerously low level within a few minutes by the inhalation of 1% Fluothane vapour. On occasion a slow respiratory rate is seen, but more usual is a tachypnoea associated with a reduction in the minute volume. As the concentration of Fluothane vapour is increased, so the respiratory rate rises and the minute volume falls, giving a reduced tidal exchange (Fig. 1). This is made manifest by cyanosis when Fluothane and air are administered to the patient but, in the presence of oxygen, the colour will remain good even though the respiratory exchange is inadequate.

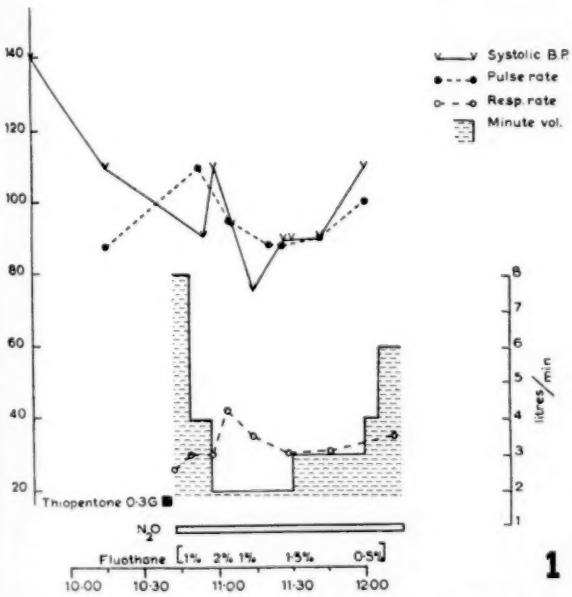

Fig. 1. Prostatectomy. Age 52. Premedication with 1/100gr. of atropine. The minute volume varies with the concentration of Fluothane vapour. As the minute volume falls, so the respiratory rate increases. The pulse rate and blood pressure tend to follow each other and are also influenced by changes in the concentration of Fluothane.

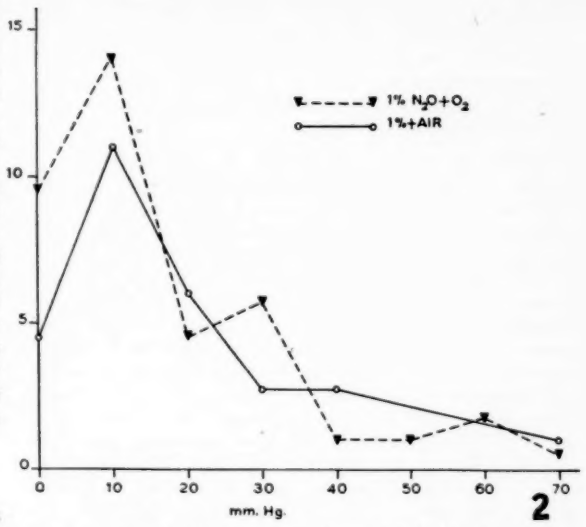

Fig. 2. Graph showing the maximum fall in blood pressure (systolic) after inhaling 1% Fluothane vapour for 5 minutes. Ordinates indicate the numbers of subjects in each group.

It is imperative that this effect should be fully understood by anyone contemplating the administration of Fluothane. A method of inflation must always be available and respiration must be assisted on the least suspicion. Failure to do so will inevitably lead to a tragedy.

The depression of minute volume is related to some extent to the pre-operative medication that the patient has received. No more than 50 mg. of pethidine combined with 1/100 gr. of atropine, or the atropine alone, should be used, since heavier sedation will depress respiration still further.

Effect of Fluothane on the Cardiovascular System

Johnstone[7] has made an extensive electrocardiographic study of the effects of Fluothane on the heart. He regards Fluothane as a 'safe' anaesthetic agent in this respect, but insists that atropine should be given as premedication to avoid extreme sinus bradycardia, which otherwise may be encountered. A degree of bradycardia almost always occurs, but arrhythmias are not to be expected.

Hypotension is common, but is rarely extreme unless a high concentration of Fluothane is administered. The blood pressure tends to fall within the first few minutes of an anaesthetic, but thereafter remains constant unless it is influenced by additional factors or by raising the concentration of Fluothane. As a rule the general condition of the patient remains surprisingly good, and with the general appearance and strong pulse which can be felt at any artery, it is difficult to believe that any appreciable degree of hypotension is present. No variation was noticed in the blood pressure fall with Fluothane and air as compared with

Fluothane, nitrous oxide and oxygen (Fig. 2). In both instances the pulse rate and the blood pressure tended to follow each other closely, and on this account atropine may be recommended as a means of moderating a too rapid fall of blood pressure. In more extreme instances Johnstone recommends the use of Vasoxyl.

Fluothane and Muscle Relaxants

The use of suxamethonium in conjunction with Fluothane anaesthesia is not remarkable and the results are identical with the use of this drug during any other anaesthetic.

Gallamine tri-iodide has been used in doses up to 120 mg. and on occasion there has been a slight fall in blood pressure. However, since the blood pressure and pulse rates under Fluothane tend to be parallel, there may be a slight rise in blood pressure probably associated with the tachycardia so commonly seen when gallamine is used in other circumstances.

With d-tubocurarine the effect on the cardiovascular system tends to be more dramatic, and falls in blood pressure are seen more commonly. They are often profound, particularly when the concentration of Fluothane exceeds 1% v/v. It would thus appear that suxamethonium and gallamine may be used during Fluothane anaesthesia with safety, but that d-tubocurarine should be employed only with caution, if at all. Neostigmine must also be considered in relation to the muscle relaxants, and its effects are of equal significance. Under Fluothane anaesthesia a much more profound fall in pulse rate is likely to be seen than usual, and this may be associated with a calamitous degree of hypotension. Such a response can be avoided by giving a large dose of atropine, at least 1/75–1/50 gr., 5 minutes before an injection of neostigmine. Neostigmine must then only be given in small divided doses at a rate not exceeding 1 mg. per minute, with 2·5 mg. as the maximum dose.

Recovery

Recovery from Fluothane anaesthesia is usually rapid but, naturally enough, will be influenced by any pre-operative drugs that may have been administered and any other supplementary medication. Apart from these, consciousness is usually regained within about 10 minutes of stopping the administration, provided that the patient has an adequate respiratory exchange. Recovery is characterized by a surprisingly rapid return of mental alertness and also, since Fluothane has little analgesic effect, the early appreciation of pain. In children this often leads to restlessness, which is best treated by the administration of an analgesic drug.

Nausea and vomiting are less than after most anaesthetics, and the patients commonly state that they feel fit. In a few instances, bouts of shivering may occur before full consciousness returns. These cannot be explained, but they may be provoked by painful stimulation.

DISCUSSION

Fluothane has many advantages, of which non-flammability, smooth and rapid induction, simple methods of administration, and quick recovery without undesirable side effects, are the most important. To these may be added freedom from irritation of the respiratory passages, the apparent absence of damage to the liver and kidneys, and often a bloodless operating field. But these claims must be balanced against depressed respiration, hypotension, bradycardia and the cost of the drug. The significance of the effects on the heart are not yet fully appreciated, nor are the mechanisms by which the changes in respiration and circulation are brought about. However, serious depression of respiration, even at light levels of anaesthesia, must be regarded as a warning against the injudicious use of the drug in unskilled hands.

It might have been hoped that Fluothane could replace chloroform in domiciliary practice; but where an open drop technique is chosen it is unlikely that efficient means of resuscitation will be immediately to hand. Also, since domiciliary practice implies obstetrics, one further difficulty remains. Although as an anaesthetic for the mother Fluothane has proved satisfactory, convenient and safe, yet it does pass the placental barrier and produces a fall in the foetal heart rate, often commensurate with the maternal bradycardia. Thus the obstetrician may be faced with the dilemma of deciding whether a slow foetal heart rate is due to foetal distress or to Fluothane.

CONCLUSION

Fluothane has proved to be an ideal agent for the induction of anaesthesia, and I regard it as being superior to ethyl chloride not only in children but also in adults. It can also be used in preference to trichlorethylene in a nitrous oxide and oxygen sequence because of the wider range of anaesthesia it produces and the ease with which the depth of anaesthesia may be altered. For major surgery requiring complete upper abdominal relaxation, I do not think that any real advantage can be claimed, although different techniques and methods of use might well alter this view.

With adequate precautions the open drop method of administration is satisfactory, although the criticism may be raised that the concentration of vapour administered is unknown. The same criticism applies to any open technique, but with our present limited experience of such a powerful agent as Fluothane it is probably justified. To be consistent, though, the administration of Fluothane from a vaporizing bottle which cannot be calibrated accurately and permanently must also be condemned,[8] especially as such pieces of apparatus as the E.M.O. and the Fluotec are available and efficient in use. Fluothane is not supposed to undergo any decomposition in the presence of soda lime; but the employment of a closed-circuit technique, even though economical, carries the risk of a dangerous concentration of the drug being reached in the system. An overdose may thus be given in a short space of time and already one tragedy has been reported from this cause.[9]

Finally, I would express the opinion that Fluothane is a valuable anaesthetic drug, but that its use, particularly by the inexperienced anaesthetist, should be limited and cautious. It would be a great pity if an agent with so many potentialities gained an evil reputation through misuse and misunderstanding of its properties before it had received a fair trial.

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FUNKSIONELE GESLAGTELIKE AFWYKINGS

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Gedurende 'n algemene praktyk van 8 jaar het slegs twee mans na die skrywer gekom met die klagte van onmag of impotensie, en dit wil dus voorkom dat onmag nie so dikwels by die man voorkom nie. Die statistiek by die vrou oor dieselfde tydperk was egter heeltemal anders. Ruim 90% van die vroue was geslagtelik onmagtig; omtrent 70% het nooit orgasme bereik nie voordat hulle nie meer ervare was nie of kinders gehad het nie. Omtrent 15% verloor die vermoë tot orgasme ná kindergeboorte. Omtrent 10—15% was normaal. Die verskil in geslagtelike vermoë tussen mans en vroue is ontsettend.

By die naslaan van die gesaghebbende literatuur merk 'n mens 'n verbasende toestand van sake op. 'n Erkende mediese woordeboek se beskrywing van hierdie toestand meld skaars die vrou; skynbaar raak sy alleen by organiese onmag betrokke. 'n Gesaghebbende mediese ensiklopedie meld glad nie die vrou in sy relaas oor onmag nie. Die teksboeke oor die ander vertakkings van die medisyne, met inbegrip van dié oor die ginekologie en obstetrie, het òf niks nie òf baie min oor hierdie toestand te sê.

Dit is miskien verstandig om eers ons definisies te verklaar. Ons definisie van *onmag* is 'n mislukte orgasme, as gevolg van die een of ander oorsaak. *Orgasme* beteken ‚intense opgewondenheid, veral soos dit plaasvind gedurende geslagtelike omgang, en wat gevolg word deur gieting van die semen by die man, of ontspanning en *detumescence* by die vrou'. *Dyspareunia* beteken pynlike of moeilike geslagtelike omgang, en kan op niks anders as impotensie uitloop nie. *Frigiditeit* beteken 'n verlies van geslagtelike drang en dit kan ook op niks anders as onmag uitloop nie. *Libido*, of geslagtelike drang, beteken ook psigiese energie. Dit is duidelik uit hierdie definisies dat onmag verreweg die algemeenste klagte is ten opsigte van geslagsgemeenskap. Dit is in hierdie opsig dat die dokter die meeste daarmee te kampe het, en dan ook baie meer dikwels by die vrou as by die man.

Sir Alan Herbert skryf in ‚Holy Deadlock': ‚If the bedroom's not right, then every room in the house is wrong.' Dit is ongetwyfeld waar, en het betrekking op alle verhoudings tussen man en vrou. Die geslagtelike lewe is die spil waarom die lewes van die meeste van ons draai. Geslagtelike drang of libido ontwikkel nie oornag by die jongeling nie. Dit begin reeds by die suigeling, en ontwikkel gaandeweg, en speel deur die hele lewe lank 'n groot rol. Dit projekteer selfs weg van die teenoorgestelde geslag op dinge—plekke en diere is voorbeelde.

By die vrou verleen die geslagtelike lewe en die voortplanting betekenis aan haar hele bestaan. Geslag word vandag baie vrymoedig bespreek in die pers, oor die radio, in die teater en op straat. Ons is meer bewus daarvan en van ons geslagtelike probleme. Ons statistiek dui daarop dat die vrou meer pynlik bewus is van haar onmag as die man van syne. Miskien praat en skrywe ons te veel daaroor en prikkel ons die gedagte, terwyl daar nie 'n ooreenstemmende geleentheid geskep word vir die uiting van die drang nie. Hierdie onbalans gee miskien aanleiding tot baie van ons probleme, waarvoor 'n oplossing gesoek moet word.

Gemeenskap is die finale stap in die geslagtelike lewe. Die vrou gaan wesenlik in haar maat op by hierdie kritiese moment—anatomies, fisiologies, biologies en sielkundig. Hierdie akte word bekroon met orgasme. As die orgasme nie bereik word nie, is die vrou, volgens ons definisie, onmagtig. Gemeenskap is 'n komplekse handeling. Die aanleidende oorsaak of aansporing moet in die brein gesoek word. Indien verstandelike aanleiding ontbreek, kan normale gemeenskap nie plaasvind nie. En net soos die normale coitus dáár sy oorsprong het, het die mislukte gemeenskap ook sy oorsprong in die brein.

Wat gebeur as die vrou impotensie ervaar? Slapeloosheid, senuspanning, 'n argwaan teenoor die gemeenskap, dyspareunia en frigiditeit kom dikwels voor. Masturbasie word soms die uitvlug. In dié opsig verg masturbasie besondere aandag. Kragtens ons definisie is *masturbasie* die opwek van 'n orgasme deur die eie hantering van die geslagsorgane. Dit staan ook bekend as eie-misbruik. Hoewel ons natuurlik eens is met die eerste van hierdie twee benamings, stem ons hoegenaamd nie met die tweede een saam nie. Dit kom baie in die laer vorms van die lewe voor en verskaf plesier en verligting. Dit kom heel vroeg by die mens voor—by die baba en jong kind—en natuurlik dikwels by die jongeling om en by die puberteit. Dit dien 'n normale natuurlike doel, maar vanweë omgewing, opvoeding en die geheimsin wat geslagsake omhul, het die mening posgevat dat masturbasie iets uit die bose is. In die dierelewe is daar geen skuldgevoel daaraan verbonde nie. In die beskaafde menselewe is dit ook 'n natuurlike verskynsel. Die konvensie bring egter mee dat 'n groot persentasie mense op 'n sekere stadium skuldig begin voel na die beoefening van masturbasie. Daar is hoegenaamd geen ernstige gevaar daaraan verbonde nie. Die mens wat ná masturbasie skuldig voel, het in die eerste plek die handeling uit die verkeerde oogpunt benader, want die meeste mense wat hierdie metode van verligting toepas vind baat daarby sonder enige verdere reaksies.

Die impotente vrou gaan egter nie op 'n normale wyse oor tot masturbasie nie. Dit kom alte dikwels voor wanneer die geslagsgemeenskap eers misluk het, en dien dan as uitvlug. By masturbasie word die vulva en die vagina geprikkel, maar in besonder is die clitoris en nymphae gevoelig. Cooke maak die volgende stelling: ‚With the continued stimulation by friction of the sexual nerve-endings, the sexual centre in the lumbar cord becomes excited to the point of sending out the stimuli which result in orgasm—the crisis of intercourse.' Daar is reeds daarop gewys dat die gedagte wat aanleiding gee tot beide coitus en masturbasie sentraal in sy oorsprong is.

Wat gebeur as ons die vroulike reaksie nagaan wanneer die clitoris masseer word by 'n geval van onmag? Die gewone vaginale ondersoek soos voorgeskrewe in die teksboeke maak byna nooit melding van die toets van die vulva-vaginale sensiwiteit nie. Dit sou verstandig wees van die geneesheer om altyd as hy 'n vaginale ondersoek doen, ook

die gevoeligheid te toets en te let op die reaksie van die pasiënt wat kla van onmag.

Ruim 50 dergelike gevalle is ondersoek. Die pasiënt word vooraf ingelig dat die ondersoek as toets dien. Haar vertroue moet gewen word; sy moet ontspan en meewerk en boweal eerlik wees met haar antwoorde. Nadat die roetine ondersoek van inspeksie, palpasie ens. voltooi is, word eers die posterior vagina-wand liggies geprikkel. Nie een enkele pasiënt het dit opwindend gevind nie. Dit het by 90% pyn veroorsaak, en 10% het geen gevoel gehad nie. By beweging van die uterus in 'n posterior-anterior of laterale rigting het 50% gesê dat 'n aangename sensasie opgewek word, en die ander 50% het dit as 'n ‚aaklige gevoel' beskryf. Waar daar 'n organiese afwyking was, spreek dit vanself dat pyn die reaksie sal wees, maar ons bespreek hier die funksionele gevalle. By die masseer van die urethra het 10% van ons pasiënte 'n aangename gevoel ondervind; 10% geen gevoel nie, en 80% het dit as onaangenaam beskryf. By die masseer van die clitoris het 5% dit as onaangenaam beskryf, maar by 95% het dit 'n aangename gevoel opgewek. Van hierdie 95% behou ongeveer 10% die aangename sensasie net vir 'n rukkie en daarna het hulle geen gevoel daar nie. Die orige 85% was daarvan oortuig dat volgehoue prikkeling op orgasme sou eindig en dat hulle dan die gemeenskap met sukses sou kan voltooi.

GEVOLGTREKKINGS

Ons het tot die volgende gevolgtrekkings gekom:

1. Die man is selde onmagtig.
2. 90% vroue is onmagtig.
3. By onmag en ander funksionele geslagtelike klagtes moet die vaginale sensitiwiteit altyd getoets word by die roetine ondersoek van die vagina.
4. By die gevoeligheidstoets het ons gevind dat ruim 50%—80% impotente vroue wel die vermoë tot suksesvolle coitus besit.
5. Die man moet in alle gevalle aangeraai word om sy orgasme so lank moontlik te beheer, en beide die man en die vrou moet in dergelike gevalle spesiale onderrig ontvang ten opsigte van die verstandige tegniek van coitus, wat só moet plaasvind dat daar voldoende massering van die gevoeligste areas is. Daar moet by sulke gevalle 'n lang periode van voorbereiding en geslagtelike prikkeling wees voordat daar oorgegaan word tot die werklike akte van gemeenskap.

THE PROFESSION OF MEDICINE AND THE MODERN WELFARE STATE

WITH SPECIAL REFERENCE TO THE PRESENT DIFFICULTY IN THE UNITED KINGDOM *

S. WAND

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This is a problem which is facing every country in the world. It is not a new problem. It is a problem that has been growing in magnitude at an accelerating rate over the past few years and continues to grow.

It was said a couple of centuries ago that you could measure the civilization of a country by the way it treated its poor. We have advanced a long way from that conception. Social security or the Welfare State is concerned with everybody; with the prevention and cure of disease, care of the young, nutrition, provision of payments during periods of sickness, retirement pensions, maternity allowances, and so on. As most of these benefits can only be provided through a nationally organized scheme, politics must play a big part—so far as medicine is concerned most doctors think too big a part.

ORIGINS

The Welfare State would appear to be a product of the second World War, but its roots go much deeper. Before the beginning of this century it was realized that much of the poorer section of the population could not afford even the most urgent medical care, and 'friendly societies' grew up which encouraged people to pay a small weekly subscription. The societies in their turn made contracts with the doctors for the care of their members at so much per week, quarter or year—the beginning of the capitation-fee system. In many areas doctors, individually or in co-operation, operated similar schemes. For the very poor there were the poor-law doctors who provided service on a part-time basis, usually at an annual salary. This was charity, and the working men, when sick, often had to fall back on this form of charity. Wages were low and a period of unemployment or sickness found them with no alternative but to accept charity. Most doctors in private practice were prepared to waive the fee in many of these cases, but in the slum areas in times of industrial depression it could be impossible for them to do this for obvious economic reasons. The hospitals played an important part but they did not provide domiciliary attendance. Maternity cases were often left to the handy woman with no medical training or qualification. It goes against the grain for a doctor to see a human being suffering without medical aid simply because there is not the money to pay for it. Many patients were too proud to accept charity.

A demand grew up for some kind of compulsory insurance which would provide sick-pay. Thus was born the Welfare State. In Great Britain, Lloyd George produced his Insurance Act; the basis of payment—part by the man whilst in work, part by the employer, and the remainder by the State—is the principle in operation in Great Britain to-day. The scheme at its inception included the lower-paid workers only (and not all of these).

But when you pay out sick benefits you need a certificate of unfitness for work and this you can only get from a doctor. When you go to the doctor you have to pay a fee, and when a workman is sick he may not be able to afford a fee. So the doctor had to be brought into the scheme. Thus began an association between doctor and State in the field of general practice, an association, partly at any rate, the result of the need for certificates. As each certificate is a cheque on sick funds, there was a call for devices to prevent laxity in certification. Diagnoses needed to be divulged. Even today, in some countries, this aspect of medical secrecy has not yet been fully resolved.

It has, indeed, been said that careless certification can be such a charge on the National Treasury that it can jeopardize a social-security scheme and that some degree of control is therefore necessary of those responsible for providing the certificates.

Pensions were also paid to old people, cash benefits to the unemployed and, in the present Welfare State, various other items such as children's allowances, subsidized foods for children,

* An address given at a meeting of the Natal Coastal Branch of the Medical Association of South Africa on 9 September 1957.

school meals, maternity allowances, retirement pensions, educational grants, and so on. With most of these no one would disagree. The children must be cared for, and the sick, and the old and infirm. Nevertheless, there can be no doubt that too extensive application of the principle of social security has taken away a measure of incentive to be self-supporting. The individual has come to rely far too much on the State to make every provision for him. The State is to make him secure (if in these days there can be security). He is apt to forget that he is the State, he has to make the ultimate provision, he has to learn to use the benefits judiciously if they are to go round.

Let us look for a moment at the hospitals. In the United Kingdom the hospitals were originally founded on charities and until quite recent times depended upon charitable bequests—except the Poor Law hospitals. Medical treatment made many advances, particularly in the field of surgery, and hospital treatment began to cost more. The standard of nursing and hygiene improved and this cost money. In the course of time Poor Law hospitals were turned into municipal hospitals paid for out of the rates. The voluntary hospitals, as the others were called, found that they could no longer continue on private benefaction even though their non-resident medical staff gave their services free, and contributions were asked for from the workers, who gave, at first, 1d. per week through contributary schemes.

MEDICAL PRACTICE IN THE WELFARE STATE

When the War ended, the British Government asked Sir William Beveridge, as he was then, to draw up a report on social insurance and this Beveridge Report was the blue print for the Welfare State in the United Kingdom. By this time many changes had taken place. Medical science had made prodigious strides. The cost of medical care had increased enormously. It needed much more personnel and equipment. The lives of the people being prolonged, there was a higher proportion of old people to be cared for, which meant more money out of the National Exchequer. Killing diseases became chronic diseases, the victim being often unable to work. At the same time changes had taken place in incomes. Income tax was levied in such a way as to have a considerable effect in the lowering of the higher incomes so that there was a much smaller disparity in net expendable income between the lowest paid worker and the highest paid executive. The result of this, coupled with the greatly increased cost of medical care, was that the number of those who could afford to make their own arrangements was much smaller. Hospital costs bounded up and up. The cost of essential modern drugs is prohibitive to most people in an illness.

No doctor will disagree with the maxim that the aim of medical practice is to deny nobody such medical care as is necessary to save his life, cure his illness or alleviate his suffering, and in practically all countries today that means an overriding responsibility for the provision of medical care, which can only be achieved by the intervention of the State, at any rate in some part. But how? How can this be achieved whilst at the same time retaining for the public and the profession that liberty and confidence which are fundamental to a proper doctor-patient relationship? It is not too much to say that most of the countries of the world have been floundering towards a solution. In many cases too much was attempted too quickly. The problem is to find a Health Service adequate in content, fair in application with no one denied for financial reasons, affording the best medical care, yet with an unharrassed and contented medical profession, having proper incentives and maintaining their proper status in the community.

A prolonged period of under-employment or under-production would have the most serious repercussions in the Welfare State. The raising of the school age and the increasing proportion of old people mean greater demands on the Welfare funds. The same factors reduce the number of those on whose efforts the size of the Welfare Fund depends.

Full employment and maximum production are therefore essential to the successful Welfare State. These factors produce consequential results in the field of medicine. In the first place the medical service must be so efficient and so easily available that the worker is not left unproductive for longer than necessary. As the doctor, by his certificate, determines the fitness of a person for work and the payment by the State of sickness benefit to the worker and his family, he is a most important person, indeed a key person, in keeping up production. It can be understood how great may be the temptation for the politician to endeavour to get control.

A policy of full or over-full employment means an inflationary trend. Industry and the worker have the means of keeping in step with inflation; so has the independent self-employed person. Not, however, those who are remunerated directly by the State, for every claim is immediately met with the cry, 'No increase! It will start an inflationary chain'.

The provision of welfare benefits by the State is a popular plank in political platforms. In the field of medical care budgets can be made of their cost, but these may easily under-estimate the considerably increased use that will be made of the medical services and the inevitably increased cost of the constant advances being made in medical science. These increased costs can be a severe embarrassment to the Government, for once the public has been accustomed to the provision of a medical service with little or no direct payment, it is very difficult indeed for any political party to bring about any contraction in the services provided. Economies must be found somewhere. The costs of treatment and the remuneration of the doctors can be challenged—how much more easily in a full-time State-controlled salaried service?

State help may mean State control. If wholly dependent on the State for remuneration the doctor and the practice of medicine are brought very close to the political economy. Because of his peculiar vocational position the doctor can be singled out for poor treatment or an intransigeant attitude. For these and other reasons doctors all over the world have been wary and suspicious, and in the World Medical Association meetings, medical problems concerned with social security have played an important part in the discussions.

PRINCIPLES OF THE WORLD MEDICAL ASSOCIATION

The World Medical Association has drawn up certain principles of social security and medical care. Briefly, in general terms, these are as follows:

Right of patient to choose his doctor and doctor his patient.

No third party to interfere between patient and doctor.

If the service is to be controlled, the control should be exercised by the doctors.

Patient to be able to choose his hospital.

Freedom of doctor to choose his place and type of practice.

No restriction of medication or mode of treatment.

Appropriate representation of doctors on official bodies dealing with medical care.

It is not in the public interest that doctors should be full-time salaried servants of Government or social security bodies.

In a social security or insurance plan any doctor to be at liberty to participate or not.

Compulsory health insurance plans should cover only those unable to make their own arrangements.

Remuneration of medical services ought not to depend directly on the financial condition of the insurance organization.

To this I would add the freedom to publish and to criticize.

Overriding all is the Hippocratic Oath.

These principles are regarded as fundamental to the best practice of medicine. Like all general principles they are to be interpreted with common sense; obviously a woman would not be able to choose to go to an eye hospital for her confinement. The underlying truths are sound. Most of them are so platitudinous that it would seem unnecessary to mention them, yet a close study will show how easy it is for them to be transgressed in a controlled service.

The basis of all medical care is a satisfactory doctor-patient relationship. The patient places himself in the hands of the doctor, confident that his doctor will do everything possible for him, and that no one will be able to interfere in that care. The secrecy of the consultation is sacred.

The doctor is prepared to devote his life, irrespective of hours of work or his family responsibilities, to the care of his patient. There can be no greater responsibility given to any man than that of having the life of another human being in his hands.

In a fully controlled service a patient might not be able to choose his doctor; a doctor might be directed where to practise; the form of treatment he has to carry out might be determined by others; he might have no say in the organization of the service;

he might have no right to publish or to criticize. He might even be compelled to divulge information received in consultation.

When the State becomes the sole paymaster the financial status of the profession is at the whim of the State, a very important matter in an inflationary economy. The doctor cannot refuse to treat patients. He cannot go on strike.

In a free-enterprize medical service a doctor will advance by his knowledge, skill, industry and personality and the patients will critically evaluate his worth. In a State-controlled service his advancement will be in the hands of others. It may be a lay, medical or mixed body, but other factors may enter. When lay bodies have had the appointment of doctors to posts in their hands, it has not been unknown for nepotism or political bias to determine the issue. At the inception of such a service the profession may be satisfied that the terms of service are satisfactory and that the full freedom of the profession is maintained, but if this freedom becomes restricted the nature of his calling and his specialized training tie the established doctor. If he dislikes working for the State or if he regards his rewards as inadequate he has not the same opportunities as others for escape. Indeed, he does not wish to escape. He became a doctor to do a doctor's job.

And so, once established, it may become more and more difficult for the doctor to resist encroachment by the State on his essential freedoms, and this is particularly the case where part or whole of his remuneration takes the form of salary. The need for the profession to be watchful, prepared and united is obvious. Once control is established it is easy to tighten the screw.

It is also possible for the State, by the use of the University grant and the subsidy to the student as well as by amendment of the law governing the criteria of admission to the Medical Register, to effect considerable increases in the number of those qualified to practise medicine.

It is of the utmost importance, therefore, that when it is found necessary for the State to play some part in the provision of medical care it shall be in such a way that the doctor can still carry out his work with freedom in the way he thinks best, that the doctors' statute shall not be reduced, and that the relationship of full confidence between the patient and the doctor shall not be jeopardized.

BRITISH NATIONAL HEALTH SERVICE

In the National Health Service in Great Britain there is free choice of doctor and patient, and the doctor in the Service has the right to practise where he wishes (except in over-doctored areas—a small number of areas determined by an independent committee, mostly medical). The doctor has full freedom in treatment and the right to publish and criticize. The profession is well represented at all levels in organization committees and there is the fullest consultation before any changes are made in the terms and conditions of service. There is also the most complete regard for the ethics of the profession.

Let us look briefly at the alternative methods of giving medical care in a Welfare State. The financial help of the State can range from a contribution to the upkeep of the hospitals to a fully organized service. The patient can be left to pay his own fees or make an independent insurance arrangement if it is available. The State can encourage such an insurance arrangement by itself making a contribution; the insurance can be made compulsory. In one country the patient makes a voluntary insurance contribution, the State makes a corresponding payment and it is left to the patient to pay the remainder. In this instance the State also provides free of charge what are called life-saving drugs.

In Great Britain everyone of working age has to pay a weekly contribution, a similar contribution being paid by the employer (or the contributor if he is self-employed). Only a small proportion of this money, however, goes to the Health Service, the bulk being used for other social security benefits. The cost of the Health Service is met very largely from general taxation monies. Before the Acts were passed, there was considerable discussion whether the Service should apply to everybody or should exclude those who were willing and able to make their own arrangements—what was called the 90% issue.

When the State pays for the whole of the Health Service, it can make some arrangement with the doctors or organize a full-time salaried service. I need hardly say that in the United Kingdom we are opposed to a full-time salaried service. I do not think that I need go further into the reasons for this.

How can the doctor be remunerated in a State-paid service? For the presiding hospital staff, for those in the public health service of the local authority, and for research workers, it must be by salary. The hospital consultant and specialist staff can be given the option of being on a full-time or part-time salaried basis with the right to private practice, beds for private patients being available for their use in hospitals under agreed conditions.

For general practitioners there are three principal alternative methods of remuneration:

1. *By salary.* This must presuppose a full-time salaried service. It must contravene many of the principles I have referred to previously. It is interesting to speculate whether, the services given being similar, a full-time salaried service would not prove more expensive than one provided in the more traditional ways.
2. *By capitation fee*, with or without a limitation in lists and a right to private practice. The doctor gets paid so much a year for each patient on his list. If the capitation fee is the same for all doctors, his only incentive is extension by increase in the number of patients.
3. *By items of service.* This was tried on a very limited scale in the early days of the National Health Insurance and was found to be subject to abuse.

In Great Britain there can be no doubt that the Service has conferred great benefits on the public, clearly shown by the use that has been made of it. The economic barrier having been removed, every member of the public can receive the fullest and most up-to-date medical care; every pregnant woman is entitled, without payment, to the care of her own doctor, midwife, consultant or hospital as required; domiciliary consultant services are available without charge and many peripheral hospitals have been up-graded (making skilled consultants and specialists more readily available). The local authorities provide ambulance service, a home-nursing service, a home-help service to give domestic help in homes when women are confined and, when available, in cases of sickness or for the old and infirm; and many other after-care and welfare services.

The doctors, having decided to enter the Service, have made every effort, as you would expect, to make it a success. The public has come more and more to rely on the doctor for every aberration from the normal and is increasingly seeking advice for the preservation of normal health and for the prevention of illness. It was expected that a free service would lead to an increase in the work of the doctor. It certainly has done so. We accept that, but we expect to be reasonably treated so far as our status and remuneration are concerned.

THE PRESENT DISPUTE IN THE U.K.

Before we came into the Service certain committees known as the Spens Committees, were set up to determine the proper range of remuneration of the doctor with due regard to the desirability of maintaining in the future his proper social and economic status. The findings of these committees were accepted by the Government and the profession. We claimed, after the Service came into being, that these findings were not being translated into remuneration, having regard to the changed value of money and certain other factors. Our contention was upheld in 1952 by Mr. Justice Dankwerts in what came to be known as the Dankwerts Award.

As money depreciated the value of the award diminished. We asked for the difference to be made up. We used legal and moral arguments to support our contention. The Government refused to accept or to arbitrate on them. That was the dispute. The Government decided to set up a Royal Commission to compare the remuneration of doctors with that of other professions, to make recommendations for a range of remuneration, and possibly for a way to deal with such disputes in the future.

Subsequently, statements were made and promises given which, in fact, modified the terms of reference of the Royal Commission. A small interim adjustment in remuneration was made and we agreed to give evidence to the Royal Commission and await its findings.

The dispute, however, brought other anxieties to a head. In the hospital field there is a bottle neck. Many registrars with full consultant qualifications and training, although they have reached

the late thirties, cannot get consultant posts. Most of us think that there is need for an increase in the number of consultants. These registrars are necessary for the efficient working of the hospitals and in many cases they do a consultant's work. There are not enough vacancies and it is very difficult indeed for these men and women, at such a late age, often with family commitments, to carve out a career in any other branch of medicine.

Then again, it was confidently expected that the general practitioner would, in the new Service, come closer to the hospital by taking an increasing part in the work of the hospitals and by the increase in the number of general-practitioner beds. This has not proved to be the case. It is now almost impossible for a general practitioner (or a consultant) to change his type or place of practice once he is settled. And, finally, there is not sufficient incentive to the general practitioner.

The British Medical Association has decided that now that the Service has been in existence for nearly 10 years it is necessary to take a critical look at all its aspects, and we are appointing a committee with strong lay (i.e. consumer) interest to consider it.

CONCLUSION

This is a very big subject and I have tried to look at it from a wide angle so far as is consistent with the title. I have indicated some of the pitfalls, dangers and difficulties. The need for a Welfare State and the means used to provide it will depend upon the genius, desires and material welfare of the people of the State, but wherever the practice of medicine is involved the liberty of the doctor relative to his work, the preservation of his status in society—so necessary to his work—and his ultimate responsibility to the patient alone, remain paramount. It will behove the doctors of the State to be vigilant and united to this end.

EVALUATION OF GRIP LOSS

Abstract of Article by* JOHN E. KIRKPATRICK, M.D.

San Francisco

In assessing the degree of permanent disability after a hand injury, the loss of grasping power is a measurable factor and is therefore of value. In this article, which is prepared by the author for the Committee on Industrial Health and Rehabilitation of the California Medical Association, the various factors involved in weakening the grip, and the various methods of accurately measuring the grip loss are considered.

An instrument for accurately measuring comparative loss of grasping power is known as a dynamometer. There are three types, each incorporating a different principle:

The Geckeler dynamometer is a pneumatic instrument which records the compression of a rubber bulb (a rolled-up blood-pressure cuff may be used in a similar way). The method, however, is generally inaccurate and unreliable as a quick jerk of the hand on the cuff may give almost any reading.

The Collins dynamometer incorporates an oval spring, the compression of which activates a pointer across a dial. However, the lack of uniformity of the spring, and the discomfort which it causes in the hand on using the instrument, discourages the patient from gripping as strongly as he might and makes the instrument inaccurate and unacceptable.

The third type is the Jamar dynamometer, which is unanimously recommended by the committee. It incorporates a sealed hydraulic system, and differs from the previous two types in that it measures grip *force* and not grip *pressure*. This is important since two hands with similar functional grasps will show different grip *pressures* if they are of different sizes, since in the larger hand the same force is spread over a larger area. On the other hand the *grip force* is not dependent on the size of the hand, nor is it affected if one or more fingers are amputated. This instrument therefore records a more accurate measurement of relative function.

Loss of grasping power is measured as a percentage of the estimated normal for that hand. In estimating this normal, the uninjured opposite hand is used as the basis for comparison, the grip in the major hand being arbitrarily assumed to be 10% greater than in the minor hand. For example, if the uninjured hand is the minor hand and its dynamometer readings average 100 lb., then the injured major hand's estimated normal should be 110 lb. If the dynamometer reading in this hand is, however, only 70 lb. then the percentage grip loss in this injured major hand is 40/110 or 36%.

Even an accurate dynamometer can be misleading under certain circumstances, however. Errors may arise owing to confusion as to which is the major and which is the minor hand, while to be accurate the dynamometer presupposes that the normal extremity is in fact perfectly normal and that the extremity under assessment was normal before the injury under review. Any pre-existing abnormality in either the normal extremity or the extremity under assessment invalidates the calculation, as of course does any failure on the part of the patient to cooperate with his best efforts. The dynamometer assessment should therefore be considered in the light of a careful clinical evaluation of all the factors which cause grip loss, for in practice any functional impairment of the hand can affect its grasping power. Amputaf tions of fingers and thumb, limitations in joint movements of the fingers and thumb, pain, and muscular weakness or incoordination, may all be factors in reducing the grasping power, and in practice more than one of these disabilities is usually found to be operating in any single case.

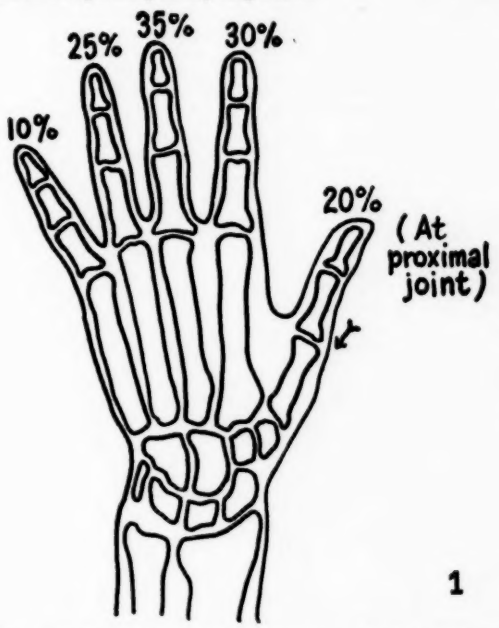

Fig. 1. Estimated approximate percentage of grip loss due to amputation of entire finger. For partial loss of any finger: approximate loss at middle joint—$\frac{3}{4}$ value of finger; approximate loss at distal joint—1/5 value of finger. (Kirkpatrick, J. E. (1957): Industrial Medicine and Surgery, **26**, 287—by permission.)

* From *Industrial Medicine and Surgery* (1957), **26**, 285. Published at the request of the Workmen's Compensation Commissioner, Pretoria.

The approximate grip loss which may be anticipated after various levels of amputation in the fingers and thumb is illustrated in Fig. 1, while limitation of finger and thumb movements also bears relationship to grip loss, and although this ratio is not reliable, it is of value in calculating the grip loss which may be anticipated, from which may be deduced whether the case is ready for final assessment or not.

Pain in the hand, the wrist, the elbow or the shoulder is another factor which may affect the grip of the hand, and its importance may be evaluated by obtaining (1) a description of the activity which produces the disability, (2) the duration of the disability, (3) the activities which are precluded and those which can be performed with disability, and (4) the means for relief. *Pain* as a disabling factor may also be graded into degrees of severity as follows:

(*a*) a severe pain would preclude the activity precipitating the pain.

(*b*) a moderate pain could be tolerated but would cause marked handicap in the performance of the activity precipatating the pain.

(*c*) a slight pain would cause mild handicap in the performance of the activity precipitating the pain.

(*d*) a minimal pain would constitute an annoyance but would not interfere with activity.

Muscular weakness will also result in grip loss and is usually associated with muscular wasting. Most persons doing active work have a greater girth on the major side, but the difference may vary from almost nothing in a frail female worker to nearly an inch in the girth of the arm of a strong male. Equal bilateral measurements of girth in active persons usually indicates some atrophy of the major side but a small decrease in girth on the minor side is usually not significant. Measurements of the girth of the arms and forearms of both injured and uninjured extremities should therefore be recorded.

The coordination of muscular action is an important factor in developing an adequate grip and therefore any failure of fixation of the wrist by the extensors, as in tenosynovitis or radial nerve palsy etc. interferes markedly with the normal grip.

To summarize, therefore, in examining any upper extremity for assessment purposes the following information is necessary:

1. The girth of the arms and forearms on both sides, as evidence of muscular wasting and muscle weakness.
2. Measurement of joint movements and the distances in inches between the tips of the fingers and the distal palmar crease after the fullest finger flexion possible.
3. A description of any pre-existing defect or grip loss in either the injured or uninjured extremity.
4. A statement as to which is the major hand.
5. Dynamometer readings (with the Jamar dynamometer) of the injured and uninjured sides, the average of three readings being taken.
6. A description of the original injury, the course and duration of the treatment and the end result.

T.L.S.

NIERBESERINGS *

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Nierbeserings is betreklik seldsaam (minder as ·05% van alle toelatings by 'n algemene hospitaal) maar is belangrik vanweë 'n groeiende toename as gevolg van vinniger verkeer en die uitbreiding van meganisme in die boerdery en nywerheid, omdat kinders dikwels die slagoffers is, en omdat daar, ten spyte van die erns van die besering, met deeglike behandeling 'n klein sterftesyfer is en 'n siektetoestand selde nagelaat word.

Patologiese Anatomie

Weens die veilige ligging van die niere diep in die buikholte geniet hulle 'n mate van beskerming en word hulle, met min uitsonderings, alleen beseer by strawwe trauma. Direkte trauma kneus of verpletter die nier teen die laaste rib of teen die werwels, b.v. wanneer die wiel van 'n voertuig oor die liggaam loop. Die feit dat die niere 'n mate van beweeglikheid het vrywaar hulle verder van drukbesering. Aan die ander kant veroorsaak die beweeglikheid soms besering as gevolg van 'n sweepslag-beweging, soos by 'n val van 'n hoë afstand af.

Daar kinders geen perinefriese vetomhulsel ontwikkel tot ná puberteit nie, word hulle niere makliker beseer en kom skeuring van die peritoneum ook voor.

Die trauma wat nierbesering veroorsaak is nooit gering nie, en meegaande besering soos geskeurde lewer of milt en frakture van ribbes en werwels kom dikwels voor. Ons moet veral op ons hoede wees as geringe trauma gevolg word met tekens en simptome van nierbesering. Dit dui 'n onderliggende patologiese toestand van die betrokke nier aan. 'n Hidronefrotiese nier, of 'n nier vol stene, word baie makliker beseer as die normale orgaan.

Die twee vernaamste komplikasies van nierbeserings is bloeding en urien-uitsyfering.

Indeling

Nierbesering wat op direkte of indirekte trauma volg heet *toebeserings*. Dié wat veroorsaak word deur steek- of koeëlwonde is *oopbeserings*. In die aard van die saak rig laasgenoemde wonde gewoonlik minder skade aan in die nier. Nogtans vereis sulke wonde in die reël 'n laparotomie omdat ons hier te doen het met 'n penetrerende buikwond en besering, veral van die dermkanaal, moet met sekerheid uitgeskakel word. Steek- of koeëlwonde wat die nier tref is veral gevaarlik en belangrik vanweë 3 moontlikhede, nl.:

(*a*) Direkte besering van die niersteel met massiewe bloeding.

(*b*) Direkte besering van die bekken of ureter met lekkasie van urine.

(*c*) Die moontlikheid van 'n wond aan die dermkanaal.

Om dié redes is 'n laparotomie binne die eerste paar uur na die ongeluk gewoonlik nodig, en 'n buiktoegang word aanbeveel. Waar die pasiënt se toestand dit toelaat en die geriewe byderhand is, is dit wenslik om by nierbeserings van dié aard 'n binneaarse piëlogram te neem voordat die laparotomie gedoen word. Dit verskaf dikwels waardevolle inligting van nierfunksie of uitsyfering sodat die besluit op nefrektomie of ureterhegting vergemaklik word nadat die toestand van die ander buikorgane vasgestel is.

Wat *toebeserings* van die nier betref ken ons verskillende grade van besering, nl.: (1) kneusing, (2) skeuring, en (3) verbrokkeling. En as seldsame moontlikhede kan dit gebeur dat die niersteel of die ureter skoon afgeruk word.

BEHANDELING VAN TOEBESERINGS

1. *Kneusing*

Hier word die nier sodanig beseer dat die parenchyma gekneus word sonder dat die nierkapsel skeur. Daar vind dus geen bloeding buite om die nier plaas nie. Daar is egter dikwels 'n skeur in die kelk, want makroskopiese haematurie kom gereeld voor. Die pasiënt toon gewoonlik min skok; daar is haematurie (wat kan wissel indien stolsels die ureter of bekken verstop) en daar is gereeld spierspanning en pyn in die lende. Geen massa is in die niergebied voelbaar nie. Die bloeding duur gewoonlik 'n paar dae en klaar dan op.

Binneaarse piëlogram wys min veranderings. Daar is wel soms belemmerde funksie. Binneaarse piëlogram is vir elke geval wenslik, nie soseer om die trauma te bevestig nie as om vas te stel dat daar wel twee gesonde niere is. Dit moet altyd onthou word dat abnormale niere makliker beseer word as die normale orgaan. Retrograadse piëlografie is selde nodig om die diagnose te bevestig. Word dit gedoen sal dit dikwels die skeuring van die kelk aandui.

* 'n Referaat gelewer by die Stigtingsvergadering van die Afdeling Kalahari, Tak Wes-Kaapland, Upington, 31 Augustus 1957.

Die behandeling is konserwatief. Die pasiënt moet ten minste 10-14 dae rus—verkieslik in die bed. Te vroeë beweging en veral energieke inspanning kan sekondêre bloeding veroorsaak wat baie lastig kan wees en afbreuk doen aan die verwagte goeie prognose. Dit kan selfs nefrektomie nodig maak.

Die prognose by kneusbesering van die nier is goed. Daar behoort geen siektetoestand van die nier te volg nie. Meegaande beserings kan egter die prognose heelwat beïnvloed.

2. *Nierskeuring*

Dit word deur strawwer trauma veroorsaak en gaan meer dikwels gepaard met beserings van die milt en lewer, beenfrakture ens. wat grotendeels daartoe bydrae om die sterftesyfer hoër te hou. Hier is daar wel skeuring van die kapsel met uitsyfering van bloed en dikwels ook urien in die renale fossa. Behalwe die simptome en tekens soos by kneusing is daar ook gereeld skok, groter bloedverlies, en gewoonlik 'n massa in die lende te voel.

Die behandeling word makliker saamgevat as daar twee afsonderlike kliniese groepe erken word, nl.:

(*a*) *Nierskeuring met buikkomplikasies.*

(*b*) *Nierbesering sonder tekens van ander buikorgaan-besering.*

(i) Daar dit so dikwels gebeur, veral by kinders, dat die nierbesering nie so erg is nie, is dit 'n probleem wat spesiale aandag verdien. Ons moet hier rekenskap hou met die moontlikheid van lewer-, milt- of dermbesering en nierbesering daarby. Daar is dikwels toenemende tekens van inwendige bloeding en hier word dit gewoonlik besluit om 'n laparotomie te doen onderwyl skok en bloedverlies met bloedoortapping beheer word. Daar is in sulke omstandighede geen tyd om piëlografie te doen nie. Die nierbesering in sulke gevalle is baie keer van sekondêre belang, in elk geval totdat die buik oopgemaak is en die nodige behandeling aan ander organe gegee is. Word dit dan gevind dat die nierbesering ernstig is met veel plaaslike bloeding en uitsyfering, sal 'n nefrektomie gedoen word, maar eers *nadat* die aanwesigheid en normaliteit van die teenoorgestelde nier deur ondersoek en met betassing vasgestel is. Word daar nie veel skade in die een of ander niergebied gevind nie, dan word die buik daarna toegemaak, en die niertoestand word met X-straalondersoek later noukeurig vasgestel.

'n Belangrike les wat hier geleer word is dat die urien altyd by buikbeserings ondersoek moet word vóór die laparotomie gedoen word. Dit is 'n onnodige en onaangename verassing om 'n groot haematoom in die niergebied te vind omdat daar versuim is om die urine vóór die operasie te ondersoek.

(ii) In die kliniese groep nierbeserings waar beserings van ander buikorgane nie vermoed word nie, word daar in die eerste plek behandeling gegee vir skok en bloedverlies. Die gewone nierskeuring sal na verloop van 48 uur sodanig herstel het, dat binneaarse piëlografie gedoen kan word. Dit gee kennis van die aanwesigheid van een normale nier en 'n aanduiding van hoedanig die ander een beseer is. Funksie is gewoonlik swak en uitsyfering word selde met hierdie ondersoek bespeur. Waar daar die minste twyfel bestaan is retrograadse ondersoek nodig. *Dit is belangrik om vas te stel of die geval net kneusing is en of daar 'n skeuring van die kapsel met die moontlikheid van urien-uitsyfering is.* Waar dit wel gebeur het moet die nier ondersoek word en òf geheg, òf verwyder word. Hoe vroeër na die ongeluk hierdie besluit gemaak word, hoe beter is die kans om die nier te red. Nietemin is dit selde moontlik om goeie binneaarse piëlogramme te verkry voor skok beheer is en nierfunksie herstel het. Daar is dus gewoonlik 'n vertraging van 2-3 dae na die ongeluk voor hierdie besluit geneem word.

As die kliniese beeld dié van 'n skeuring met uitsyfering van bloed en urien in die renale fossa is en word dit met binneaarse en retrograadse piëlografie bevestig, dan is nefrektomie by die meeste gevalle die veiligste, die gouste en die beste behandeling. Met ander woorde, as die besluit gemaak word om die nier per operasie te ondersoek dan volg nefrektomie gewoonlik. Dit word nie aanbeveel om 'n geskeurde nier te probeer herstel en red as die pasiënt se lewe op die keper is nie.

3. *Verbrokkeling van die Nier*

Hierdeur bedoel ons 'n verpletterde nier met veelvoudige skeure deur die kapsel en die kelke, en gewoonlik ook deur die omhullende vet en fascia.

Weens die skok, bloeding en toenemende massa in die lende is dit gewoonlik vroeg duidelik dat nefrektomie nodig is, en dikwels in dié gevalle is nefrektomie die enigste manier om die bloeding te keer. Waar die besering duidelik en hoofsaaklik die nier getref het, is 'n lende-toegang gebruiklik. Wanneer daar besering van ander buikorgane vermoed word, sal 'n buiktoegang gewoonlik verkies word.

Alhoewel sulke ernstige nierbeserings soms 'n noodoperasie eis, is daar in die meeste gevalle egter nog die verkieslike geleentheid om spesiale ondersoek voor operasie uit te voer. Binneaarse piëlografie wys strawwe besering met gewoonlik geen funksie nie en met retrograadse piëlografie word uitsyfering bevestig. By operasie word daar 'n groot haematoom, baie bloed en 'n verbrokkelde nier gevind. Daar is min urien aanwesig omdat die nier nie funksioneer nie.

Die hoofdoel van piëlografie is om die normaliteit en aanwesigheid van die teenoorgestelde nier te toon.

GEVOLGE VAN NIERBESERINGS

Sterftesyfer van Nierbeserings

In die ligte besering soos kneusing is die uitkoms goed en geen permanente skade aan die nierweefsel word verwag nie. Nierfunksie herstel volkome.

Die erns van nierbeserings het dikwels meer te doen met meegaande besering van ander organe. Die sterftesyfer van nefrektomie is klein en min gevalle sterwe bloot as gevolg van die nierbesering as dit vroegtydig en versigtig behandel word.

'n Niersiektetoestand na Nierbesering

Daar is baie geskrywe oor die swak resultaat van konserwatiewe behandeling van ernstige nierbeserings. Dit word beweer dat indien 'n nier herstel waar daar heelwat bloeding rondom plaasgevind het, so 'n nier vasgetrek word in digte bindweefsel met die ontwikkeling van 'n ‚Goldblatt'-nier en verhoogde bloeddruk. Die moontlikheid bestaan teoreties wel, maar word prakties selde gevind.

Dan ook sal nierbeserings met skeuring en uitsyfering wat sonder operatiewe dreinering herstel het, soms 'n onreëlmatige hidronefrose of pseudo-hidronefrose ontwikkel. Hierdie niere is natuurlik baie vatbaar vir ontsteking en stene word maklik in hulle gevorm. Sulke patologiese veranderings gebeur seer seker, maar daar is min ooreenstemming in die literatuur oor hoe dikwels dit voorkom en hoeveel skade daar veroorsaak word. Sommige is die mening toegedaan dat 80% van ernstige beseerde niere uiteindelik sodanig patologies word dat hulle verwydering vereis. Ander beweer dat alhoewel daar dikwels patologiese veranderings plaasvind, die pasiënt min las daarvan het, goeie nierfunksie behou en maar selde verwydering van die betrokke nier nodig het.

Dit kom my voor of ons dit moet aanvaar dat daar wel gevaar bestaan om 'n ernstige beseerde nier te laat sit, en dat waar die ander nier normaal is, dit verkieslik is om so 'n nier vroegtydig te verwyder, tensy dit met chirurgiese blootlegging maklik en met veiligheid herstel kan word. Met ander woorde, elke nier wat ernstig beseer is (geskeur of verpletter) word verkieslik met operasie ondersoek. Selfs met operasie is dit dikwels moeilik om te besluit of die nierweefsel lewensvatbaar is. Indien die geskeurde nier geheg word en die gebied sorgvuldig dreineer word is die gevaar van uitsyfering met die moontlikheid van fibrose en pseudo-hidronefrose minimaal. Die gevaar van ontsteking en veral sekondêre bloeding word egter nie vermy nie en beide van hierdie komplikasies mag later nefrektomie verg. Dit is my mening dat waar daar twyfel bestaan, dit veiliger is om radikaal te wees en nefrektomie te doen mits die teenoorgestelde nier normaal is.

SAMEVATING

Nierbeserings word behandel namate die erns van die besering. Kneusing word met min uitsondering konserwatief behandel en die pasiënt moet ten minste 10-14 dae rus; piëlografie dien as die maatstaf van diagnose en herstel.

Kritieke beserings met verbrokkeling, niersteel-besering en ernstige bloeding wat nie beheer kan word nie, vereis 'n spoedige nefrektomie.

Die middelklas beserings met skeuring van die kapsel gee gewoonlik tyd om die probleem volledig met behulp van X-strale deeglik vas te stel. Duidelike uitsyfering uit die kelksisteem vereis in die meeste gevalle nefrektomie. Dit is soms moontlik om die nier met hegting en dreinering te bly behou veral waar die ander nier bedenklik is.

RECENT EXPERIENCES WITH POLIOMYELITIS VACCINE

JAMES GEAR

Poliomyelitis Research Foundation, Johannesburg

The value of poliomyelitis vaccine was reviewed at the Fourth International Poliomyelitis Congress recently held in Geneva by the authorities concerned from the United States of America, Canada, Britain, Denmark, France, Sweden, Australia, and South Africa. In each of these countries the vaccine has been used on a large scale and a wide experience of its use has been accumulated. As there apparently is still some doubt in the minds of the medical profession and, more particularly, in those of the lay public, about the safety and value of this vaccine, it may be worth while to summarize the general conclusions.

In the United States, which has had far greater experience than any other country, the programme of immunization of children and, more recently, of adults up to the age of 40, is being steadily implemented and well over 100,000,000 doses of vaccine have been issued. This vast experience has confirmed that the vaccine is safe and that there is a notable reduction in the incidence of paralytic poliomyelitis in vaccinated children as compared with unvaccinated children of the same age-group. The protection ranges from 75 to 90% in those inoculated with 2 or 3 doses of vaccine.

In Canada the low incidence rate of poliomyelitis during the past 2 years has made evaluation of the results of the vaccination programme somewhat difficult, since the number of reported cases in both vaccinated and unvaccinated groups was rather small. In 1955 there were 5 cases of paralytic poliomyelitis in 600,000 vaccinated children and 51 cases in 890,000 unvaccinated children of the age-group 5-9. In 1956 there were 11 cases of paralytic poliomyelitis in 1,860,000 vaccinated children and 136 cases in 2,140,000 unvaccinated children of similar age-groups. These findings, although the incidence of poliomyelitis is at an exceptionally low level, are in agreement with the more extensive experience in the United States.

In Australia approximately 2,200,000 of the 2,500,000 children under 15 years of age have received one or two injections. The incidence of poliomyelitis reported for the 1956-7 season is much lower than for any year in the past decade. Although further experience will be necessary before the conclusion can be fully justified, it is felt that what appears to be a 90% reduction in incidence is primarily a result of immunization.

In Britain, in a large-scale trial carried out in 1956, it was found that the incidence of paralytic poliomyelitis in the vaccinated children was about one-fifth of the incidence in unvaccinated children.

In Denmark over 90% of individuals under 16 years old have been vaccinated and it is not possible to get comparative reports, but the incidence of poliomyelitis during the last 3 years is the lowest in the last 2 decades.

In Israel, all children between the ages of 6 months and 3½ years have been vaccinated, a total of approximately 130,000. The vaccine was administered intracutaneously in two 0·3 ml. amounts spaced 1 month apart. The third inoculation will be given this winter. Although no comparative data are yet available, a notable reduction in the incidence of paralytic poliomyelitis has been observed as compared with previous years.

It will not be possible to assess accurately the value of the vaccine in protecting against paralytic poliomyelitis until about 20 years have elapsed since its introduction. However, these figures, obtained from all the countries using vaccine on a large scale, indicate clearly that the vaccine has been of value in the period covered by the observations in reducing the incidence of paralytic poliomyelitis significantly.

It is also clear that some limitations of this type of vaccine have become apparent. The protection conferred does not prevent alimentary infection. Indeed, vaccinated children have been shown to acquire alimentary infection as readily as unvaccinated children, and to excrete the virus in abundance for as long a time as unvaccinated children. This type of vaccine therefore presumably will not result in the elimination of virulent poliovirus from the community. This may be expected to continue to circulate and to cause epidemic waves of infection during which the 'normal' proportion of the unvaccinated children will be liable to develop paralytic poliomyelitis. As with any killed vaccine, full protection is not given by the formalinized type of poliovirus vaccine and a few paralytic cases in fully vaccinated children may also be expected to occur.

It is also clear that one dose of vaccine given to a large number of children during an epidemic will not hasten that epidemic's end. The likelihood of the occurrence of coincidental cases at such time, of course, is greatly increased. However, there is little evidence that the inoculation of poliomyelitis vaccine has any provoking effect in precipitating paralysis in children who otherwise would have had a silent infection, and it is generally agreed that vaccination could be continued with advantage during an extensive epidemic.

To summarize, the evidence is clear that vaccine prepared according to the prescribed method and tested according to the prescribed safety and potency tests, is safe and of considerable value in preventing paralytic poliomyelitis.

BOOKS RECEIVED : BOEKE ONTVANG

Surgery Principles and Practice. Edited by J. Garrott Allen, M.D., Henry N. Harkins, M.D., Ph.D., Carl A. Moyer, M.D., Jonathan E. Rhoads, M.D., D.Sc. With 26 contributors. Pp. xxii + 1495. 621 Illustrations. £5 10s. 0d. net. London: Pitman Medical Publishing Co., Ltd. 1957.

Die Vaskulären Erkrankungen im Gebiet der Arteria Vertebralis und Arteria Basialis. Von Prof. Dr. H. Krayenbühl und Dr. M. G. Yasargil. VIII + 170 Seiten. 125 Abbildungen in 205 Einzeldarstellungen. DM 77.—. Stuttgart: Georg Thieme Verlag. 1957.

Orthopaedic Surgery in Infancy and Childhood. By Albert Barnett Ferguson, Jr., B.A., M.D. and five other contributors. Pp. XII + 508. Price 120s. London: Baillière, Tindall & Cox Ltd. 1957.

The Year Book of Endocrinology, (1956-57 *Year Book Series*). Edited by Gilbert S. Gordan, M.D., Ph.D., F.A.C.P. Pp. 377. $6·75. Chicago: Year Book Publishers, Inc. 1957.

The Year Book of Neurology, Psychiatry and Neurosurgery—1956-57 *Year Book Series*). Edited by Roland P. Mackay, M.D., S. Bernard Wortis, M.D. and Oscar Sugar, M.D. Pp. 596. $7·00. Chicago: Year Book Publishers, Inc. 1957.

Modern Medicine for Nurses. 4th Edition. By Patria Asher, M.D., M.R.C.P. Pp. xiii + 378. Figs. 99. 22s. 6d. net. London: William Heinemann Medical Books Ltd. 1957.

Aids to Materia Medica and Therapeutics. Fifth Edition. By J. W. Hadgraft, F.P.S., F.R.I.C. Pp. vii + 259. 10s. 6d. London: Baillière, Tindall and Cox Ltd. 1957.

Anaesthesia and Otolaryngology. By Donald F. Proctor, M.D. Pp. xv + 267. Figs. 33. $7·00. Baltimore: The Williams & Wilkins Co. 1957.

The Closed Treatment of Common Fractures. Second Edition. By John Charnley, B.Sc., M.B., F.R.C.S. Pp. xii + 256. 199 Figures. 25s. net + 11d. postage abroad. Edinburgh: E. & S. Livingstone Ltd. 1957.

Medical Treatment. By Kenneth MacLean, M.A., M.D., F.R.C.P. Pp. vii + 696. 50s. net. London: J. & A. Churchill Ltd. 1957.

Progress in Gynecology. Volume III. By J. V. Meigs, M.D., and Somers H. Sturgis, M.D. Pp. xii + 780. $15·50. New York and London: Grune & Stratton Inc. 1957.

The Practice of Industrial Medicine. Second Edition. By T. A. Lloyd Davies, M.D., F.R.C.P. Pp. vii + 282. 15 Figures. 30s. net. London: J. & A. Churchill Ltd. 1957.

ASSOCIATION NEWS : VERENIGINGSNUUS

DISTRIKSGENEESHERE-VERENIGING-JAARVERGADERING

Die Jaarvergadering van die Distriksgeneeshere-Vereniging is op 21 September 1957 in Durban gehou. Namens die Departement van Volksgesondheid was Dr. J. J. du Pré le Roux, Dr. H. F. Schiller en Dr. J. D. Gertenbach teenwoordig, asook die volgende distriksgeneeshere: Drs. D. J. Serfontein, R. Cloete, L. Lawson, G. R. Masey, N. Gilliland, J. A. du P. Kriek, L. J. Botha, T. M. Crouch, C. Riekert, P. Spaarwater en G. F. C. Troskie.

Fooie

Daar is verslag gedoen dat 'n vermeerdering van fooie vir bevallings, chirurgiese fooie en medisyne-toelaes, waar die betrokke geneesheer statistiek kan lewer, deur die Tesourie toegestaan is, tesame met vermeerdering van sekere fooie vir ondersoeke met sertifisering en immunisering ens. Dit is van krag sedert Oktober 1956.

Die Voorsitter het sy verslag gelewer en het die Sekretaris van Gesondheid en Dr. Schiller bedank vir hulle aandeel in die verkry van beter besoldiging vir distriksgeneeshere. Hy het ook besonderhede in verband met die nuwe Memorandum van Ooreenkoms en die farmakopia genoem.

Verklaring deur die Sekretaris van Gesondheid

Die Sekretaris van Gesondheid het sy teleurstelling uitgespreek met die gebrek aan belangstelling by distriksgeneeshere in hulle eie sake—slegs 10 uit 400 het die Jaarvergadering bygewoon. Hy het die vele onnodige klagtes en die gebrek aan kennis van die werking van die hele aangeleentheid aan hierdie gebrek aan belangstelling gewyt.

Hy het gemeld dat 67 distriksgeneeshere 'n salarisverhoging ontvang het aangesien hulle statistiese bewys gelewer het van die omvang van hulle werk. Hy het ook melding gemaak van die opfrissingskursusse vir distriksgeneeshere wat nou weer ingestel is en deur die Departement gesubsideer word. Die eerste een wat in Pretoria gehou was, was 'n groot sukses.

Dr. Schiller, die assistent-sekretaris, het die volgende sake onder die aandag gebring. Die nuwe vorm vir die aanvraag om vervanging van spesiale medisynes sal binnekort uitgestuur word aan distriksgeneeshere, en die farmakopia sal binnekort gedruk word. In verband met chirurgiese fooie het hy gesê dat daar baie onnodige korrespondensie is oor klein operasies waar normaalweg geen ekstra fooi betaal word nie, en wat distriksgeneeshere tog eis.

Na bespreking is dit besluit dat geneeshere gevra word om, waar 'n operasie nie gemeld word in die amptelike lys nie, die betrokke geneesheer 'n verduideliking van die omvang en presiese aard van die operasie saam met sy eis te stuur, sodat die geneesheer wat die rekeninge nagaan, in staat is om te besluit of 'n fooi wel geregverdig is al dan nie.

Lykskouings in die Veld

Die vergadering was dit eens dat, as gehandel word in ooreenstemming met die omsendbrief insake lykskouings, die saak primêr dié van die polisie is en dat die geneesheer slegs 'n professionele getuie is. As dit dus nodig is om 'n lykskouing te hou, moet dit waar moontlik in 'n lykhuis onder goeie omstandighede gedoen word om van volle waarde te wees.

Tuisbehandeling van Tering

Die verteenwoordigers van die Departement van Volksgesondheid is spesiaal bedank vir hulle welwillende hulp in verband met die tuisbehandeling van tering.

Geskenk aan die Liefdadigheidsfonds

Die sekretaris het die finansiële staat ingedien. Die inkomste aan ledegelde het £88 10s. 0d. beloop, en die uitgawe vir die jaar was £101 12s. 7d. Daar is 'n opgehoopte kapitaal van £255 15s. 3d. Lede van die Distriksgeneeshere-Vereniging het 'n bedrag van £357 aan die Liefdadigheidsfonds van die Mediese Vereniging geskenk as dankbetuiging vir die verhoogde fooie. Dit is besluit dat, nadat alle verdere bydraes ontvang is, hierdie som aangevul sal word uit die opgehoopte kapitaal om die totaal op £500 te staan te bring.

Nuwe Bestuur

Die nuwe bestuur is as volg saamgestel—*Voorsitter:* Dr. D. J. Serfontein. *Sekretaris:* Dr. G. F. C. Troskie. *Komitee-lede:* Drs. L. J. Botha, J. A. du P. Kriek, C. Riekert, P. Spaarwater en T. M. Crouch.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

APPROVED MEDICAL AID SOCIETIES

MEDICAL AID SOCIETIES

The following list of approved medical aid societies is published for general information. Members are requested to keep this list for reference because it no longer appears in the tariff book. After each meeting of the Federal Council an up-to-date list is published in the *Journal*, including societies that have been newly approved and omitting those that have been withdrawn.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
21 October 1957

1. A.A. Mutual Medical Aid Society, P.O. Box 9595, Johannesburg.
2. Abercom Group Sick Benefit Society, P.O. Box 715, Port Elizabeth, Cape Province.
3. African Cables Medical Benefit Fund, P.O. Box 172, Vereeniging, Transvaal.
4. African Explosives Medical Aid Society, P.O. Box 1122, Johannesburg.
5. African Homes Trust Sick Fund, P.O. Box 93, Cape Town.
6. African Oxygen Limited Medical Aid Society, P.O. Box 5404, Johannesburg.
7. Afrikaanse Pers Beperk se Siekefonds, Posbus 845, Johannesburg.

GOEDGEKEURDE MEDIESE HULPVERENIGINGS

MEDIESE HULPVERENIGINGS

Vir algemene inligting word onderstaande lys van goedgekeurde mediese hulpverenigings gepubliseer. Lede word versoek om die lys byderhand te hou want dit verskyn nie meer in die tariefboek nie. Na elke vergadering van die Federale Raad word 'n volledige lys (wat die name van pas-goedgekeurde verenigings insluit en van dié wat onttrek is weglaat) in die *Tydskrif* gepubliseer.

L. M. Marchand
Medesekretaris

Mediese Huis
Kaaptad
21 Oktober 1957

8. Alex. Aiken & Carter Medical Benefit Society, P.O. Box 2636, Johannesburg.
9. Algoa Medical Aid Society, P.O. Box 369, Port Elizabeth.
10. Anglo-Alpha (Dudfield) Medical Benefit Society, Private Bag, P.O. Lichtenburg, Transvaal.
11. Argus Medical Benefit Society (Cape Argus Branch), P.O. Box 56, Cape Town.
12. Argus Medical Benefit Society (Daily News Branch), P.O. Box 1491, Durban.
13. Argus Medical Benefit Society (Star Branch), P.O. Box 1014, Johannesburg.
14. Associated Employers Medical Aid Society, P.O. Box 7462, Johannesburg.

15. A.T.I. Medical Aid Society, P.O. Box 5057, Boksburg North.
16. Atlantic Refining Company Medical Aid Society, P.O. Box 664, Cape Town.
17. Babcock and Wilcox Medical Aid Fund, P.O. Box 545, Vereeniging.
18. Bakers Ltd. European Employees' Sick Benefit Fund, P.O. Box 692, Durban.
19. Bloemfontein Municipal Employees' Medical Aid Society, P.O. Box 288, Bloemfontein.
20. Boksburg Municipal Employees' Medical Aid Fund, P.O. Box 215, Boksburg.
21. Broderick Medical Aid Society, P.O. Box 186, Vereeniging.
22. Building Societies Joint Medical Aid Fund, P.O. Box 5728, Johannesburg.
23. S. Butcher & Sons Ltd. Medical Aid Society, P.O. Box 1004, Durban.
24. Caltex Medical Aid Society (S.A.), P.O. Box 714, Cape Town.
25. Cape Times Medical Aid Society, P.O. Box 11, Cape Town.
26. Cape Town Municipal Employees' Association Medical Aid Society, P.O. Box 1939, Cape Town.
27. Central News Agency Ltd. Medical Benefit Society, P.O. Box 1033, Johannesburg (excluding Cape Town and suburbs, Durban municipal area, Johannesburg and Witwatersrand, and Port Elizabeth and Pretoria municipal areas).
28. Chamber of Mines Medical Aid Society, P.O. Box 809, Johannesburg.
29. Civil Service Medical Benefit Association, P.O. Box 176, Pretoria.
30. Consolidated Glassworks Limited Medical Aid and Sick Benefit Society, P.O. Box 562, Germiston.
31. Corner House Insurance Fund, P.O. Box 1056, Johannesburg.
32. Coronation Medical Aid Society, P.O. Box 1517, Durban.
33. Crookes Bros. Ltd. Medical Benefit Fund, 301 Smith Street, Durban.
34. D.F.A. Medical Benefit Society, P.O. Box 610, Kimberley.
35. Eastern Province Cement Co. Ltd. Medical Aid Society, P.O. Box 2016, Port Elizabeth.
36. E.P. Newspapers Medical Aid Society, P.O. Box 1117, Port Elizabeth.
37. Egnep Medical Aid Society, P.O. Penge. Transvaal.
38. Elwamba Medical Aid Fund, P.O. Box 42, East London.
39. Escom (N.C.U.) Medical Benefit Society, P.O. Box 30, Colenso, Natal.
40. Everite Medical Aid Society, P.O. Kliprivier, Transvaal.
41. Federated Employers' Medical Aid Society, P.O. Box 666, Johannesburg.
42. Federation of Master Printers of S.A. Medical Aid Society, P.O. Box 1200, Johannesburg.
43. Ford Medical Aid Society, P.O. Box 788, Port Elizabeth.
44. Friend Medical Aid Fund, P.O. Box 245, Bloemfontein.
45. General Mining (Associated Companies) Medical Aid Society, P.O. Box 1007, Johannesburg
46. General Motors Medical Aid Scheme, P.O. Box 1137, Port Elizabeth.
47. Germiston Industries Medical Aid Society, 113 Pylon House, Human Street, Germiston.
48. Gledhow-Chaka's Kraal Sugar Co. Ltd. Medical Benefits Fund, 301 Smith Street, Durban.
49. Greaterman's Medical Aid Society (all Branches), P.O. Box 5460, Johannesburg.
50. Hollerith Medical Aid Society, P.O. Box 7018, Johannesburg.
51. Hubert Davies Johannesburg Staff Medical Aid Society, P.O. Box 1386, Johannesburg.
52. Sir J. L. Hulett & Sons Ltd. Medical Benefit Fund, P.O. Box 248, Durban.
53. Hume Cape Medical Benefit Society, P.O. Box 7, Bellville, Cape Province.
54. Hume Transvaal Medical Benefit Society, P.O. Box 204, Germiston.
55. Hunt, Leuchars & Hepburn Ltd. (Durban) Employees' Medical Benefit Fund, P.O. Box 943, Durban.
56. Hunt, Leuchars & Hepburn Ltd. (Transvaal Staff) Medical Aid Society, P.O. Box 47, Johannesburg.
57. Iscor Medical Benefit Fund, P.O. Box 450, Pretoria.
58. I.W.S. Medical Aid Society, P.O. Box 6946, Johannesburg.
59. J. W. Jagger & Co. Ltd. Medical Aid Society, P.O. Box 726, Cape Town.
60. Johannesburg Board of Executors' Medical Aid Society, P.O. Box 271, Johannesburg.
61. Klerksdorp Munisipale Werknemers Siektefonds, Posbus 99, Klerksdorp.
62. K. & L. Timbers Ltd. Staff Medical Aid Fund, P.O. Box 9, Elandsfontein, Transvaal.
63. Koegas Medical Aid Society, P.O. Koegasbridge, C.P.
64. Krantzberg Mines Medical Aid Society, P.O. Box 18, Omaruru, S.W.A.
65. Kroonstad Munisipale Mediese Hulpvereniging, Posbus 302, Kroonstad.
66. G. H. Langler & Co. Ltd. Medical Aid Society, P.O. Box 3762, Johannesburg.
67. Legal and General Medical Aid Society, P.O. Box 4870, Johannesburg.
68. Mail Times & Express Medical Aid Society, P.O. Box 1138, Johannesburg.
69. L. H. Marthinusen Medical Aid Society, P.O. Box 64, Denver, Johannesburg.
70. Masonite Medical Aid Society, P.O. Box 57, Estcourt, Natal.
71. Metal Box Company of S.A. Ltd. Medical Aid Society, P.O. Box 7752, Johannesburg.
72. Municipal Employees' Medical Aid Society (Durban), P.O. Box 625, Durban.
73. Natal Building Society Medical Aid Fund, P.O. Box 947, Durban.
74. Natal Coal Owners' (Durban Staff) Medical Aid Society, P.O. Box 281, Durban.
75. Natal Estates Sick Fund Benefit Society, P.O. Mount Edgecombe, Natal.
76. Natal Industries Medical Aid Society, P.O. Box 1300, Durban.
77. N.T.E. Staff Medical Aid Fund, P.O. Box 39, Pietermaritzburg.
78. National Industrial Credit Corporation Medical Aid Society, P.O. Box 8296, Johannesburg.
79. National Portland Medical Aid Society, P.O. Box 21, Claremont, C.P.
80. New Consolidated Gold Fields Employees' Medical Aid Fund, P.O. Box 1167, Johannesburg.
81. Northern Assurance Co. Ltd. Medical Aid Society, P.O. Box 8615, Johannesburg.
82. Northern Medical Aid Society, P.O. Box 3437, Johannesburg.
83. Northern Rhodesia Civil Servants Medical Aid Society, P.O. Box 294, Lusaka, Northern Rhodesia.
84. Norwich Union Life Insurance Society Staff Medical and Surgical Benefit Scheme, P.O. Box 1226, Cape Town.
85. Ore & Metal Medical Aid Society, P.O. Box 3548, Johannesburg.
86. Pietermaritzburg Chamber of Industries Medical Aid Society, P.O. Box 365, Pietermaritzburg.
87. Polliack Group Medical Aid Society, P.O. Box 3008, Johannesburg.
88. Pongola Sugar Milling Co. Ltd. Medical Benefit Fund, P.O. Box 194, Durban.
89. Post Office Medical Aid Society, P.O. Box 303, Germiston.
90. Pretoria Municipal Employees Sick Fund, P.O. Box 408, Pretoria.
91. Pretoria News Medical Benefit Society, P.O. Box 439, Pretoria.
92. Pretoria Portland Cement Co. Ltd. No. 1 Works (Hercules) Medical Aid Society, P.O. Box 405, Pretoria.
93. Pretoria Portland Cement Co. Ltd. No. 2 Works Medical Benefit Society, P.O. Box 7, Slurry, Western Transvaal.
94. Pretoria Portland Cement Co. Ltd. No. 3 Works (Jupiter) Medical Aid Society, P.O. Box 73, Cleveland, Transvaal.
95. Pretoria Portland Cement Co. Ltd. No. 4 Works Medical Aid Society, P.O. Box 26, Orkney, District Klerksdorp.
96. Printing Industry Medical Aid Society, P.O. Box 1993, Pretoria.
97. Prudential Medical Aid Scheme, P.O. Box 1097, Johannesburg.

98. Rand Public Service Medical Aid Society, P.O. Box 28, Boksburg.
99. Rand Water Board Sick Fund, P.O. Box 1127, Johannesburg.
100. Randles Bros. & Hudson Ltd. (Durban) Sick Benefit Fund, P.O. Box 1046, Durban.
101. Randles Bros. & Hudson Ltd. (Johannesburg) Employees' Sick Benefit Fund, P.O. Box 2678, Johannesburg.
102. Reckitt & Colman Medical Aid Society (S.A.), P.O. Box 1097, Cape Town.
103. 'Rennie' and 'The Consolidated' Employees' Medical Aid Fund, P.O. Box 1006, Durban.
104. Reunert & Lenz Ltd. Medical Aid Society (All Branches), P.O. Box 92, Johannesburg.
105. Reynolds Bros. Ltd. Medical Benefits Fund, 301 Smith Street, Durban.
106. E. S. & A. Robinson (Pty.) Ltd. Medical Aid Society, P.O. Box 293, Germiston.
107. Royal-Globe Medical Aid Fund, P.O. Box 317, Cape Town.
108. Safim Medical Aid Society, P.O. Box 223, Vereeniging.
109. Safmarine Medical Aid Society, P.O. Box 2171, Cape Town.
110. Safnit Mills Medical Aid Fund, P.O. Box 11, Jeppestown, Johannesburg.
111. Santam-Sanlam Siektefonds (Alle Takke), Posbus 1, Sanlamhof, K.P.
112. Schwartz, Fine, Kane & Co. Medical Aid Society, P.O. Box 5069, Johannesburg.
113. Shell Medical Aid Society (S.A.), P.O. Box 2231, Cape Town.
114. C. G. Smith & Co. Ltd. Medical Aid Fund, 301 Smith Street, Durban.
115. S.A. Association of Municipal Employees' (S.A.A.M.E.) Medical Aid Fund, P.O. Box 62, Pretoria.
116. S.A. Breweries Medical Aid Society, P.O. Box 1099, Johannesburg.
117. S.A.K.A.V. Sick Benefit Fund, P.O. Box 33, Paarl.
118. S.A. Mutual Fire & General Insurance Co. Ltd. Staff Medical Aid Fund, P.O. Box 516, Johannesburg.
119. S.A. Mutual Life Assurance Society Staff Medical Aid Fund, P.O. Box 66, Cape Town.
120. S.A. Press Association Medical Aid Society, P.O. Box 7766, Johannesburg.
121. S.A. Teachers' Association Medical Aid Society, 12 Bellevue Road, Sea Point, C.P.
122. S.A. Torbanite (Boksburg) Medical Aid Society, P.O. Box 5038, Boksburg North.
123. South Atlantic Corporation Medical Aid Society, P.O. Box 4610, Cape Town.
124. Southern Medical Aid Society, P.O. Box 42, Cape Town.
125. Standard Brass Medical Aid Society, P.O. Box 229, Benoni.
126. Stewarts & Lloyds Medical Benefit Fund, P.O. Box 74, Vereeniging.
127. Stuttafords Medical Aid Society, P.O. Box 69, Cape Town.
128. Sun Insurance Office Ltd. Staff Medical Aid Fund, P.O. Box 429, Johannesburg.
129. Syfret's Medical Aid Society, 24 Wale Street, Cape Town.
130. Traduna Medical Aid Fund, P.O. Box 8791, Johannesburg.
131. Transvaal Corundum Associated Asbestos Medical Aid Society, P.O. Box 72, Pietersburg, Transvaal.
132. Transvaal Society of Accountants Medical Aid Fund, P.O. Box 2995, Johannesburg.
133. U.L.A. Medical Aid Society, P.O. Box 4589, Johannesburg.
134. Umzimkulu Sugar Co. Ltd. Medical Aid Fund, P.O. Box 43, Durban.
135. United Banks' Medical Aid Society, P.O. Box 1242, Cape Town.
136. United Building Society Medical Benefit Fund, P.O. Box 7735, Johannesburg.
137. University of the Witwatersrand (Johannesburg) Staff Medical Aid Fund, Milner Park, Johannesburg.
138. Vacuum Medical Aid Society, P.O. Box 35, Cape Town.
139. Village Board of Management of Welkom Medical Aid Society, P.O. Box 708, Welkom, O.F.S.
140. Wright Boag & Head Wrightson Sick Benefit Fund, P.O. Box 183, Benoni.
141. Yorkshire Medical Aid Society, P.O. Box 2755, Johannesburg.

The following new medical aid societies were approved by Federal Council at its meeting held at Durban on 11-13 September 1957. The names are included in the above list:

1. Associated Employers Medical Aid Society, P.O. Box 7462, Johannesburg.
2. Northern Assurance Company Limited Medical Aid Society, P.O. Box 8615, Johannesburg.

Op sy vergadering van 11-13 September 1957, te Durban gehou, het die Federale Raad onderstaande nuwe mediese hulpverenigings goedgekeur. Die name is in bostaande lys ingesluit:

1. Associated Employers Medical Aid Society, Posbus 7462, Johannesburg.
2. Northern Assurance Company Limited Medical Aid Society, Posbus 8615, Johannesburg.

MEDICAL BENEFIT SOCIETIES WHICH ALLOW FREE CHOICE OF DOCTOR FOR SPECIALIST SERVICES ONLY:

MEDIESE BYSTANDSVERENIGINGS WAT VRY KEUSE VAN DOKTER ALLEEN VIR SPESIALISTEDIENSTE TOELAAT:

1. Begbie Medical Benefit Fund, P.O. Box 192, Middelburg, Transvaal.
2. Breyten Coalfields Benefit Society, P.O. Box 6, Estantia, Transvaal.
3. Broken Hill Mine Employees' Medical Specialist Fund, P.O. Box 45, Broken Hill.
4. De Beers Consolidated Mines Limited Benefit Society, P.O. Box 616, Kimberley.
5. Durban Roodepoort Deep Ltd. Benefit Society, P.O. Box 193, Roodepoort.
6. Jagersfontein Mine Benefit Society, P.O. Box 2, Jagersfontein, O.F.S.
7. Krugersdorp Municipal Employees' Medical Benefit Society, P.O. Box 101, Krugersdorp.
8. Northern Rhodesia Mine Employees Medical Specialist Fund, P.O. Box 134, Kitwe, Northern Rhodesia.
9. Public Utility Transport Corporation Sick Fund, P.O. Box 9571, Johannesburg.
10. Randfontein Estates Employees' Sick Benefit Society, P.O. Box 37, Randfontein.
11. Roodepoort-Maraisburg Municipal Employees' Association Sick Benefit Society, P.O. Box 217, Roodepoort.
12. Roodepoort-Maraisburg Non-Scheduled Mines' and Industries' Benefit Society, P.O. Box 225, Roodepoort.
13. Rosherville-Maraisburg Benefit Society, P.O. Box 99, Cleveland, Johannesburg.
14. Sasol Medical Benefit Society, P.O. Box 80, Sasolburg.
15. Simmer Pan Medical Benefit Society, P.O. Box 103, Germiston.
16. Springs Mines Benefit Society, P.O. Box 54, Springs.
17. Transvaal Jewellers' & Goldsmiths' Sick Benefit Fund, P.O. Box 8530, Johannesburg.
18. Witbank Coalfields Benefit Society, P.O. Box 26, Witbank.
19. Witbank Power Station Medical Benefit Society, P.O. Box 197, Witbank.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
18 October 1957

L. M. Marchand
Medesekretaris

Mediese Huis
Kaapstad
18 Oktober 1957

PASSING EVENTS : IN DIE VERBYGAAN

Professor R. A. Dart, head of the Department of Anatomy at the University of the Witwatersrand, Johannesburg, has been awarded the Viking Fund medal and an award of 1,000 dollars (£357) for 1957. This award is made by the Winner-Gren Foundation for Anthropological Research on the nomination of the American Association of Physical Anthropologists.

* * *

The South African Dermatology and Venereology Group. A meeting of the Cape Town sub-group will take place in the E-floor lecture theatre, Groote Schuur Hospital, on Monday 25 November 1957, at 8.15 p.m. The subject will be 'Cancer of the face'. The speakers will be: Mr. T. Schrire—Aetiology and pathology; Dr. M. B. Bennett—Radiotherapy; Mr. E. B. Malherbe—Surgical procedures; Mr. D. S. Davies—Aspects of reconstructive surgery. All are welcome.

* * *

Dr. A. Berezowski, M.B., B.Ch. (Rand), D.M.R.D., R.C.P. & S. (Eng.), voorheen van die Departement van Radiologie, Baragwanath-hospitaal, het nou by drs. E. Samuel, C. Komins en M. Denny aangesluit in hulle radiologiese praktyk te Listergebou 1, Jeppestraat, Johannesburg, in plek van dr. L. Morris wie tans oorsee vertrek het. Telefone: huis 42-9684, kamers 23-5931.

Dr. A. Berezowski, M.B., B.Ch. (Rand), D.M.R.D., R.C.P. & S. (Eng.), formerly radiologist to Baragwanath Hospital, has joined Drs. E. Samuel, C. Komins and M. Denny in consultant radiological practice at 1 Lister Building, Jeppe Street, Johannesburg, in place of Dr. Leon Morris who has left for overseas. Telephones: home 42-9684, rooms 23-5931.

* * *

The South African Society of Obstetricians and Gynaecologists, at the Annual Meeting recently held at Durban, appointed the following office-bearers: *Chairman* Dr. J. C. Coetzee, *Vice-chairman* Prof. James T. Louw, *Secretary/Treasurer* Dr. E. M. Sandler, *Committee Members* Dr. T. St.V. W. Buss, Dr. F. N. Charnock, Dr. D. P. de Villiers, Prof. J. N. de Villiers.

* * *

The South African Paediatric Association. The next meeting of the Cape Town Sub-group of this Association will be held on Tuesday, 5 November 1957, in the lecture theatre, Red Cross War Memorial Children's Hospital, Rondebosch, Cape, at 8.15 p.m. Dr. Clarence Merskey will address the meeting on 'America, Americans, and American Medicine'.

* * *

Drs. Jac. J. Theron and Hennie Pretorius, Paediatricians, 215 Lister Buildings, Johannesburg, wish to announce the following new telephone numbers:

Rooms 22-0614 and 22-0470 (not listed in directory).

Residence: Dr. Theron 41-3579, Dr. Pretorius 46-1371 (not listed in directory).

Drs. Jac. J. Theron en Hennie Pretorius, Kinderartse, Listergebou 215, Johannesburg, wil graag lede van hul nuwe telefoonnommers verwittig:

Spreekkamers: 22-0614, 22-0470 (nie in adresboek nie).

Woning: Dr. Theron 41-3579, Dr. Pretorius 46-1371 (nie in adresboek nie).

* * *

The Red Cross War Memorial Children's Hospital, Rondebosch, Cape. A meeting of the post-graduate seminar series will take place at the Red Cross War Memorial Children's Hospital, Rondebosch, Cape, on Wednesday, 6 November 1957, at 5 p.m. Dr. M. D. Bowie will speak on 'The physiology of growth', and Dr. G. Lurie on 'Heights and weights of children in the Cape Peninsula'. All are welcome.

* * *

Research Forum, University of Cape Town. The next meeting of the Research Forum will be held in the A floor lecture theatre, Groote Schuur Hospital, Cape Town, on Wednesday, 6 November at 12 noon. Dr. H. E. Schendel will speak on Some Biochemical Investigations on Kwashiorkor with particular reference to (1) serum cholesterol in clinical assessment and prognosis and (2) amino acids in the urine.

* * *

Lede word daaraan herinner dat hulle die Sekretaris van die Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, sowel as die Registrateur van die Suid-Afrikaanse Mediese Tandheelkundige Raad, Posbus 205, Pretoria, moet verwittig van enige adresverandering.

Versuim hiervan beteken dat die *Tydskrif* nie afgelewer kan word nie. Dit het betrekking op lede wat oorsee gaan sowel as dié wat binne die Unie van adres verander.

* * *

The Council for the International Organization of Medical Sciences (established under the auspices of WHO and UNESCO) has published a calendar of international congresses of medical sciences, including fixtures from the present time into 1961. A copy has been supplied to the *Journal* by the Secretary for Education, Arts and Science, Pretoria, and the Editor will be glad to reply to any enquiries concerning the calendar that may be directed to the office.

* * *

The Council for Scientific and Industrial Research. Towards the end of October 1957 the Central Administration of the C.S.I.R. will be transferred to a new site 7 miles east of Pretoria. Henceforth the President, the Vice-president and the Secretary-Treasurer, the Liaison Division and all administrative sections will be housed in the new Administrative Headquarters Building. The new telephone number is 4-5931. The postal address remains P.O. Box 395, Pretoria.

* * *

Professor Isidore Gordon, M.B., Ch.B. (Cape Town), Professor of Pathology and Dean of the Medical Faculty of the Durban Medical School, University of Natal, a member of the South African Medical and Dental Council representing this University, has recently been elected a Fellow of the Royal Society of South Africa 'in recognition of his contributions to the medico-legal field, both in respect of original work in forensic pathology as well as in his critical review of medico-legal literature'.

* * *

The Frank Forman Medical Foundation. The first award under this Foundation has been made to Dr. H. Gordon for overseas postgraduate study. Dr. Gordon is clinical research bursar in the Clinical Nutrition Research Unit supported by the Council for Scientific and Industrial Research in the Department of Medicine, University of Cape Town, where he is working on problems of coronary heart disease and its relation to diet. He hopes to leave for London next year, where he will spend 1 year in postgraduate study.

* * *

Dr. Jacques Roux, M.B., Ch.B. (Cape Town), M.R.C.O.G., formerly senior registrar, United Oxford Hospitals, and thereafter full-time gynaecologist and obstetrician, Karl Bremer Hospital, has started a consultant practice as from 1 November 1957 at 405 Medical Centre, Heerengracht, Cape Town. Telephones: rooms 3-5088, residence 97-3714.

Dr. Jacques Roux, M.B., Ch.B. (Kaapstad), M.R.C.O.G., voorheen senior registrateur, United Oxford-hospitale, en daarna voltydse ginekoloog en verloskundige, Karl Bremer-hospitaal, sal van 1 November 1957 as spesialis konsulteer te Mediese Sentrum 405, Heerengracht, Kaapstad. Telefone: spreekkamers 3-5088, woning 97-3714.

* * *

The VIIth Congress of the International Society of Haematology will be held in Rome on 7-13 September 1958. The preliminary programme will include (1) Immuno-haematology: (2) Haemorrhagic disorders: (3) Leukaemia: (4) Anaemia: (5) Neucleonics: (6) The spleen and the reticulo-endothelial system. The official languages of the Congress will be English, French, German, Italian and Spanish. The meetings will be held at the Palazzo dei Congressi, in which building the *Congress of the International Society of Blood Transfusions* will also be held on 3-6 September 1958. Further information may be obtained from Segreteria del VII Congresso Internazionale di Ematologia—Instituto di Patologia Medica—Policlinico Umberto I—Roma, Italia.

* * *

A New Medical Journal. The American Rheumatism Association announces the forthcoming publication of a new medical journal, 'Arthritis and Rheumatism', the official journal of the American Rheumatism Association, which will appear every two months starting with the January-February issue of 1958. The new journal

will cover the field of connective-tissue disorders, in particular rheumatoid arthritis, osteo-arthritis, rheumatic fever, gout, the so-called 'collagen diseases', and non-articular rheumatism. Besides original and review articles, it will include news and notices, correspondence, editorials, progress reports and book reviews. The Editor is Dr. W. S. Clark, M.D., and the publishers Grune and Stratton, Inc., New York. The address of the American Rheumatism Association is 580 Park Avenue, New York 21, N.Y., USA.

* * *

Unie van Suid-Afrika. Departement van Gesondheid. Aangifte van ernstige epidemiese siektes en poliomielitis in die Unie gedurende die tydperk 11-17 Oktober 1957.

Poliomielitis

	Bl.	*Nat.*	*Kl.*	*As.*	*Totaal*
Transvaal	3	2	–	–	5
Kaapprovinsie ..	2	–	1	–	3
Oranje-Vrystaat ..	–	–	–	–	–
Natal	1	–	–	–	1
Totaal ..	6	2	1	–	9

Pes, Pokkies, Tifuskoors: Geen.

REVIEWS OF BOOKS : BOEKRESENSIES

RORSCHACH TESTS

Rorschach Location and Scoring Manual. By Leonard Small, Ph.D. Pp. 214. $6.50. London and New York: Grune & Stratton, Inc. 1956.

Contents: Introduction. Instructions. Rorschach Location and Scoring Manual.

In 1911, Herman Rorschach, a psychiatrist, began to experiment with ink-blots, and out of these experiments there developed what has since become a widely used technique known as Rorschach Testing. The test aims at a detailed investigation of personality and the results depend upon the analysis of the candidate's interpretation of a standard set of ink-blot cards. The Rorschach is accepted as being one of the most valuable of numerous available personality tests but to become an efficient Rorschach tester requires years of specialized training and experience in the technique.

The manual under review will have meaning only to an individual who has been fully trained in the Rorschach testing. Its sole purpose is to present an indexed system of scoring according to the multiplicity of responses which can be elicited by application of Rorschach ink-blot cards. It is bound to be of very great value to the Rorschach tester.

H.C.

CANCER OF THE MOUTH

Oral Cancer and Tumours of the Jaws. A guide for the diagnosis of oral cancer and benign tumors. By George S. Sharp, M.D., F.A.C.S., F.A.C.R. (Ther.), Weldon K. Bullock, M.D., M.Sc. (Path.), John W. Hazlet, D.D.S. Plates 645. Pp. xi + 561. $15.00. New York, Toronto, London: McGraw-Hill Book Company, Inc.

Contents: Foreword by Hayes Martin, M.D. Preface. Introduction to Parts One and Two. *Part One. Introductory Principles for Neoplastic Diseases.* 1. The History of Cancer. 2. The Origin of Cancer. 3. Classifications of Oral Neoplasms. 4. The Biopsy. 5. Precancerous Pathology. *Part Two. Tumors, Benign and Malignant, of the Oral Mucosa and Accessory Cavities.* 6. Lesions of the Lips. 7. Lesions of the Tongue. 8. Lesions of the Floor of the Mouth. 9. Lesions of the Gingivae. 10. Lesions of the Cheeks. 11. Lesions of the Hard and Soft Palate. 12. Lesions of the Nasal Cavities and Paranasal Sinuses. Introduction to Part Three. *Part Three. Neoplasms, Benign and Malignant, of the Jaws.* 13. Tumors of the Epithelium. 14. Benign Neoplasms of Connective Tissue. 15. Malignant Neoplasms of Connective Tissue. 16. Neoplasms of Reticuloendothelial Tissues. 17. Lesions of Vascular and Nerve Tissue. 18. Metastatic Bone Tumors. Introduction to Part Four. *Part Four. Quasi-tumors of the Jaws.* 19. The Exostoses. 20. Fibrous Dysplasia. 21. The Reparative Granulomas. 22. Other Diseases of Bone Simulating Tumor. 23. Odontogenic Hamartomas. Introduction to Part Five. *Part Five. Cysts of the Jaws.* 24. Nonodontic Cysts. 25. Odontic Cysts. Index.

This book is exactly what the notice on its dust cover claims it to be, 'A guide for the diagnosis of oral cancer and benign tumours'. As far as this goes, the work is a comprehensive atlas of oral pathology and many of the pictures (all in black and white) are outstanding examples of the art of clinical illustration. However, in a way the book disappoints me by overemphasizing the pictures and neglecting the text. It thus defeats its own purpose and leaves the reader wanting more information.

The book deserves a place on the shelf of every surgeon, dentist and clinician dealing with oral conditions. It is too specialized for the general practitioner but is an ideal reference book which should be available in libraries for consultation by the perplexed.

The reviewer criticizes the fact that whereas many pictures of successful cases are shown, 7 or more years after diagnosis, the failures are not shown and there is no indication how many failures occur and what proportion of cases are successfully treated, so that a wrong impression can be obtained from reading this book.

This is misleading. A book on oral cancer should make it clear that in general, only the minority of cases with carcinoma of the mouth get cured. No preferred line of treatment is offered, and no indications for various forms of therapy, so that although the illustrations are wonderful, one must conclude in reflection that not one is necessary once it is accepted that a patient with a lesion of the mouth must have a proper biopsy taken and be examined by a competent pathologist.

T.S.

CORONARY ATHEROSCLEROSIS

Study Group on atherosclerosis and ischaemic heart disease: Report. *World Health Organization: Technical Report Series,* 1957, No. 117; 40 pages. Price 1s. 9d., $0.30 or Sw. fr. 1. Also available in French and Spanish. Local Sales Agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria.

This report discusses the present status of knowledge on the aetiology and pathogenesis of atherosclerosis and ischaemic heart disease and advises on means of broadening this knowledge so as to provide an eventual basis for effective prevention work.

Ischaemic heart disease is defined in the report as the cardiac disability, acute and chronic, arising from reduction or arrest of blood supply to the myocardium, in association with disease processes in the coronary arterial system. The two main pathological processes involved are atherosclerosis of, and thrombosis in the coronary vessels. Atherosclerosis includes several quite distinct intimal processes, such as fatty changes, fibrous thickening, fibrin incorporation, and calcification. In ischaemic heart disease —the end product of atherosclerosis—multiple causative factors must therefore be considered. These multiple factors may operate differently and thereby produce different pictures, in individual cases and in the disease as it occurs among various ethnic and social groups.

The main conclusion of the report is that the control and prevention of ischaemic heart disease can be brought about only as a result of improved knowledge of the relation of environmental factors and ways of life to the pathogenesis of the disease and to the consequent morbidity and mortality. The lack of information on the relation to coagulation and thrombosis of such suspected factors as genetic and environmental influences, sex, specific inborn metabolic disorders, arterial hypertension, diet (with particular reference to dietary fats), level of physical activity, stress, strain and mental tension, deserve special emphasis in research work. Possible psychological factors also need adequate study. Suggested lines of research are set out in the report, including, in an annex, a detailed description of the type of epidemiological study most likely to provide useful results. A second

annex, on public-health aspects of the disease, deals with such matters as case-finding; screening; diagnostic, social, laboratory and nutrition services; rehabilitation; and so on.

The need for the standardization of both clinical and pathological criteria and terminology in respect of ischaemic heart disease, thaerosclerosis and related conditions is regarded as sufficiently urgent to warrant the recommendation that WHO should organize a study group to undertake this task. It is also recommended that WHO should continue and expand the collection and regular publication of mortality statistics on cardiovascular and related diseases, and should consider giving assistance to national statistics services in developing the analysis of mortality by occupation and social class. Attention is also drawn to the need for improving the collection and recording of mortality data and for greater standardization of terms and procedures. Simple field studies on the basis of death certification in different countries might, it is thought, quite quickly reveal the possibilities and limitations of the international comparisons now so commonly made. Greater use of insurance company data on heart disease is advocated as an additional means of assessing the importance of the problem.

Further recommendations deal with the cooperation of FAO in studies on dietary habits and food consumption and WHO help in the training of research personnel and in various other suggested activities.

THE MOON IS FULL

The Moon is Full. By Aileen Adair. Pp. 200. 12s. 6d. net. London: Allan Wingate. 1957.

Contents: Preface. 1. I was Born in a Mental Hospital. 2. I Become a Psychiatrist. 3. The Neurosis Centre. 4. Private Practice. 5. I Go North. 6. The Lunatic. 7. The Snake-Pit. 8. The Clean-Up. 9. A Teaching Hospital. 10. Case Histories. 11. Social Life in a Mental Hospital. 12. The Law and the Lunatic. 13. A Mental Defective Colony. 14. Mental Defectives. 15. Juvenile Delinquents. 16. Escape. 17. Foreign Nurses. 18. Life Sentence without Trial. 19. 'The Mad Doctor'. 20. Out-Patients. 21. 'Physician, Heal Thyself'. Epilogue.

The glimpses which this book gives of life and conditions in mental hospitals in England is far from flattering; but then, facts seldom flatter. It is interesting reading in spite of suffering from the disadvantages that often accompany autobiography, and the author can be congratulated on her presentation of a subject which could be drab. There is hardly a facet of the life that has escaped her notice and record, and she makes some penetrating observations on the confinement of defective patients who might be usefully employed in sheltered conditions—outside an institution.

Generally it is light reading for the layman rather than the practitioner, but most doctors will find it interesting particularly if their work does not bring them into close contact with the mentally defective or disordered.

A.H.T.

THE NEW PUBLIC HEALTH

The New Public Health. Fourth Edition. By Fred Grundy, M.D., M.R.C.P., D.P.H. Pp. 214. Illustrations 21 + 23 diagrams. 18s. net. London: H. K. Lewis & Co., Ltd. 1957.

Contents: Section I. Local and Central Government. *Section II.* The National Health Service Act 1946, and Organized Medicine. *Section III.* Social Security and National Insurance. *Section IV.* The Health Department of Local Authorities. *Section V.* The Medical and Welfare Services of Local Authorities. *Section VI.* The Maternity Services and the Midwife. *Section VII.* The Prevention and Control of Infectious Diseases. *Section VIII.* The Arithmetic of Health and Disease. *Section IX.* Social Medicine and Environment. *Section X.* Other Subjects of Importance. Appendices. List of Diagrams. Lists of Tables and Graphs. List of Illustrations.

This little publication, which has been especially compiled for health visitors, midwives and social workers, attains all that it sets out to do. The fact that it has reached its fourth edition in eight years indicates only too clearly that it is fulfilling a need and has attained that popularity which it so well deserves.

Whilst the main accent is peculiar to Britain and the present organization of the national welfare state in that country, attention is also drawn to the importance of the prevention and control of all types of infectious diseases, the social aspects of medicine and to elementary vital statistics.

The author, who writes in an easy and interesting manner, has arranged his material in such a way that it is readily assimilable by even those individuals with little or no medical or nursing background.

The graphs, charts and photographs are clear and illustrative and the book is printed on very high quality gloss paper.

I have no hesitation in strongly recommending this book to persons anxious to obtain reliable and readable information of the present organization, administration and functions of the British National Health Service Act of 1946. The general message which is evident in all the chapters has a wider application and can with benefit be assimilated by medical students, midwives, health visitors and social workers in this country.

E.D.C.

CORRESPONDENCE : BRIEWERUBRIEK

LEIPOLDT-NORTIER MEMORIAL LIBRARY

To the Editor: In spite of urgent appeals which have been made through the daily newspapers for contributions towards the Leipoldt-Nortier Memorial Library Building Fund there has so far been very little response. Mostly as a result of personal appeals to friends of the late Drs. C. Louis Leipoldt and P. le Fras Nortier we have succeeded in raising £5,000, which is made up as follows:

	£
Bequest by Dr. P. le F. Nortier	1,000
Grant by Clanwilliam Municipality	1,000
Grant by Mrs. Helen Burton	500
Grant by Cape Tercentenary Foundation	500
Other contributions (including a few of £100 each) ..	2,000
	£5,000

The Memorial Library, however, with the furniture and fittings, will cost a total sum of £10,000. That means that we need a further £5,000 to carry our scheme through successfully. It will be a great blot on our generation if this undertaking has to be abandoned as a result of lack of funds. Dr. Leipoldt and Dr. Nortier were both such eminent members of the medical profession and did so much for their country and fellow men that we are under an obligation to honour them by putting up a useful monument to carry on their good work. The Library will be a fitting memorial in their honour.

An appeal is therefore made to members of the Medical Association and its different branches for contributions towards our Fund. We need help most urgently. A plaque with the names of donors of £100 or more will be put up at the entrance to the library.

Further particulars of the undertaking will be furnished with pleasure. Please address all correspondence and contributions to the Secretary, P.O. Box 4, Clanwilliam.

R. C. E. Strassberger
Secretary, Leipoldt-Nortier Library Committee

P.O. Box 4
Clanwilliam
24 September 1957

[This project took its origin in a bequest of £1,000 made by Dr. Nortier towards a library to be erected in memory of Dr. Leipoldt as one of the great figures in Afrikaans literature. After Dr. Nortier's death a committee was formed to bring the project into effect as a memorial to both Leipoldt and Nortier. It takes the form of a library building which is to be erected in Clanwilliam and which will comprise a Leipoldt room where a valuable collection of his works and items of interest from his life will be housed, a children's room, reference room, music room and a projection room. *Editor.*]